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ELECTROENCEPHALOGRAPHY, ITS CLINICAL USES AND VALUE, ILLUSTRATED FROM THE FIRST THOUSAND RECORDS AT THE ALFRED HOSPITAL.[1]

By JOHN GAME, M.D. (Melbourne),

AND

JOAN PEACOCK, B.Sc. (Tasmania),

Melbourne.

IT is proposed to discuss very briefly the principles of electroencephalography, to consider next the problem of epilepsy in moderate detail, and finally to make short reference to the use of the electroencephalogram in some other disorders. Although the purpose of this paper calls for generalizations, we have chosen to base them on our own analysis of our first thousand records at the Alfred Hospital (September 14, 1949, to June 19, 1952), from which all the examples have been drawn.

The Principles.

The altering electrical potentials of which the electroencephalogram is a record arise from the neuronal activity of the brain. Using the bipolar method as we do, the electroencephalogram amplifies differences of potential of the order of millionths of a volt between two points on the surface of the brain, as recorded through the scalp, the rhythmic variation of these potentials being their first and greatest interest. Fundamental research has shown that these rhythms do in fact arise in the cortex, but are influenced or even governed by similar rhythmical activities of deeper structures of the brain.

The following simple examples will help to make clear the grounds of these deductions and the basis of the interpretation of clinical records.

Rhythmical discharges from a dipole situated in the cortex behind electrodes equally spaced on the scalp produce waves of decreasing amplitude corresponding to the increasing distances of the electrode pairs away from the point of discharge (Figure I, A). This may be taken to represent a model of the α rhythm (8 to 14 cycles per second) in the occipital cortex.

With the origin of the discharge in the centre of a sphere, the surface of which passes through the electrodes situated as before on the scalp, the waves will have equal amplitude for all electrode pairs (Figure I, B).

With the origin of the discharge beneath one of the electrodes, the waves from the electrode pairs sharing this electrode will be out of phase (Figure I, C).

The principles underlying the other main variable, that of frequency, are more complex and less understood. In

[1] Read at a meeting of the Victorian Fellows and Members of The Royal Australasian College of Physicians, held at Royal Park on October 19, 1953.

general terms it may be stated that the slower frequencies are associated with depressed cortical activity—for example, sleep, coma, physical trauma, destructive lesions and anoxia; in the same general terms, the faster frequencies result from "activation", and the "spike" is the classical example, being usually associated with an "irritative" epilepticogenic focus, such as post-traumatic scar (Figure II).

Technique and Interpretation.

In practice we place 15 electrodes on the scalp, and a series of "runs" is recorded for several minutes each

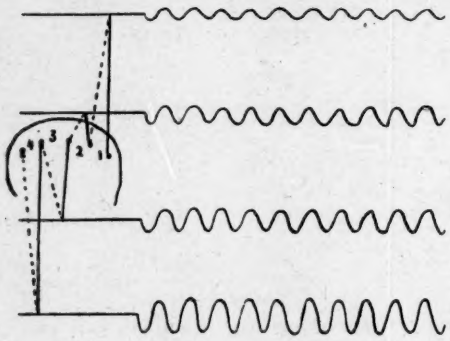

FIGURE IA.
Diagrams to illustrate the use of bipolar electrodes (Walter, 1950).

(Figure III). Our machine ("Grass" Model III*B*) records six channels in each run, and these runs may be varied at will for special purposes (Figure IV). The final record covers on the average about 100 to 120 feet of paper and takes no less than twenty minutes' continuous recording time.

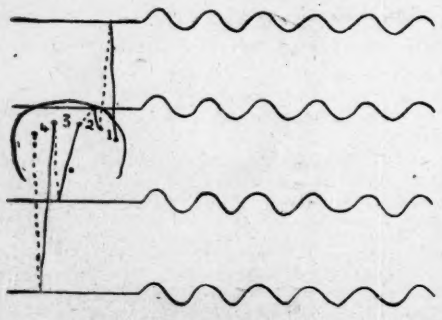

FIGURE IB.
Diagrams to illustrate the use of bipolar electrodes (Walter, 1950).

The tracing is not repetitive as in the electrocardiogram, but is continuously changing, and each record is unique. Various records of a single patient usually bear a strong resemblance to one another unless altered by structural disease—they have been likened to a person's signature.

Varying frequencies and amplitudes may appear rhythmically, irregularly, continuously, at random or paroxysmally, and may be focal, symmetrically bifocal or diffuse. The two dominant difficulties in analysis of these frequencies and amplitudes are the limitations of the human eye and the certain recognition of artefacts.

In each line of tracing there may be normally mingled about six frequencies. The possibilities of harmonics will appeal to those mathematically inclined, and the impossible task set for the human eye is obvious. Hence the evolution of the automatic frequency analyser.

Artefacts bemuse the novice and are always a bugbear. Their multiple sources in the process of amplification of such minute potentials will be well appreciated. They are too numerous to illustrate here.

The Normal Record.

The standards of normality are laid down by an International Association of Electroencephalographers. Various observers (Williams, 1941*a*; Lennox, Gibbs and Gibbs, 1940) find that 10% to 15% of normal people have abnormal records. Many of these abnormalities have no known

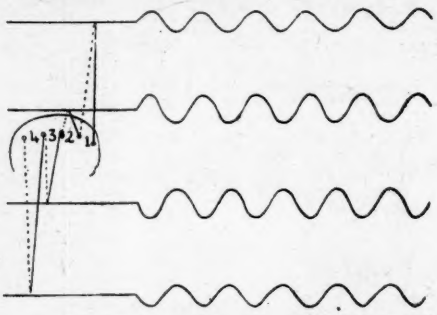

FIGURE IC.
Diagrams to illustrate the use of bipolar electrodes (Walter, 1950).

correlates. Some may denote an inherent instability (so-called dysrhythmia), often associated with epilepsy, and thus supposed to be predisposing to epilepsy in the subject or his offspring. Others may represent functional (and probably structural) immaturity. (The records of children are all grossly abnormal by adult standards, and their maturation from infancy to the early twenties is at once instructive and a source of difficulty in their interpretation.)

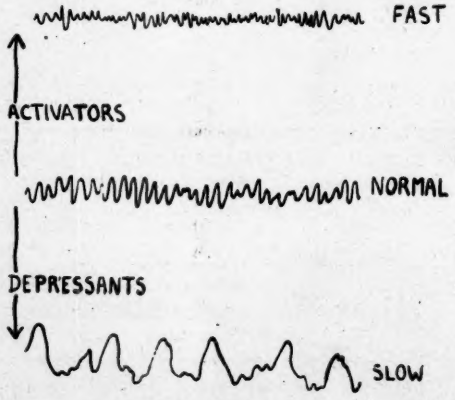

FIGURE II.
Frequency variations illustrated from actual recordings.

We have records of 91 normal people, mainly medical students and nurses. When the criteria were applied rigidly, 40% of our healthy subjects had abnormal tracings. Assessed by the "appearances" of the records with due regard to the distribution and nature of the abnormalities, we consider only 11% to be abnormal; that is, in clinical reporting we should denote them "non-specific diffuse abnormality".

We made particular observations of outbursts of δ activity (0 to 3·5 cycles per second), which were often sinusoidal, following hyperventilation (Figure VI), and

correlated in all cases with blood sugar levels taken simultaneously. The influence of the blood sugar level has an individual variation. With due regard to this, and to the ages of the subjects and the period of time between the end of the hyperventilation and the occurrence of these outbursts, many of the records would be considered epileptic by some authorities. Maybe they are; but we submit that epilepsy would cease to have a meaning as a clinical state if applied to these appearances in the electroencephalograms

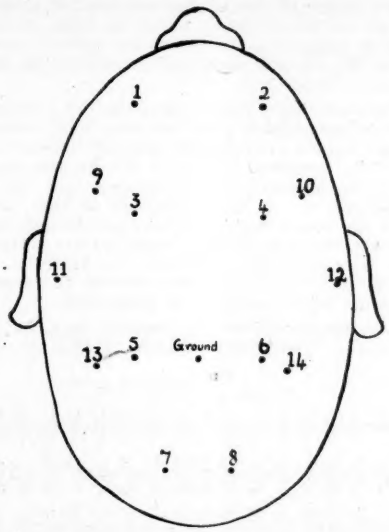

FIGURE III.
Diagram showing electrode positions on the scalp.

of healthy subjects. The final assessment of these outbursts has led us to adopt a more conservative attitude in their clinical interpretation.

Epilepsy.

The majority of epileptics can, naturally, be studied only between seizures, and the nature and significance of the abnormalities in these records will be demonstrated later.

Occasionally a seizure occurs spontaneously during recording, or can be induced by special methods of stimulation. This is most easily achieved in children who suffer "pure" juvenile *petit mal*. We have examined 524 patients in whom a diagnosis of epilepsy had been made or suspected clinically; but in only 26 of these seizures of this type occurred or were provoked. The characteristic appearance is the "wave and spike" (Figure VII). The outstanding quality of the epileptic phenomena seen electroencephalographically, as clinically, is their paroxysmal nature.

The introduction of selective drugs—for example, "Tridione"—makes it a practical necessity to separate "pure *petit mal*" from other forms of minor seizures with a clinical resemblance. Such wave and spike seizure patterns are a prerequisite to the absolute diagnosis of this condition, for which it has been suggested that the name "pure juvenile *petit mal*" should be reserved. We have been unable to distinguish these patterns from those which appear in patients who have *petit mal* associated with

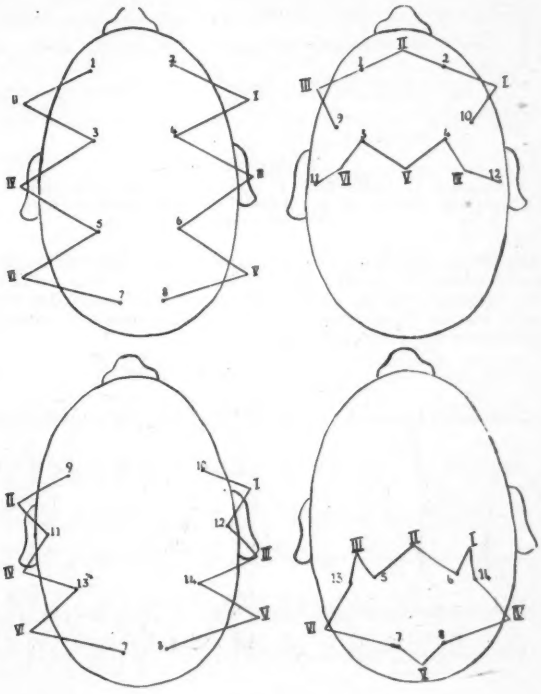

FIGURE IV.
Diagram showing four of the routine runs.

grand mal, but more elaborate methods show that these pure juvenile *petit mal* seizures have their origin not in the cortex, but in deeper central structures, and there are other differences.

Earlier studies led to the differentiation of three distinct types of epilepsy—*petit mal*, *grand mal* and psychomotor attacks—based on the different shapes of the waves in the seizure patterns. It is now becoming increasingly apparent that the form of the epileptic attacks is fundamentally

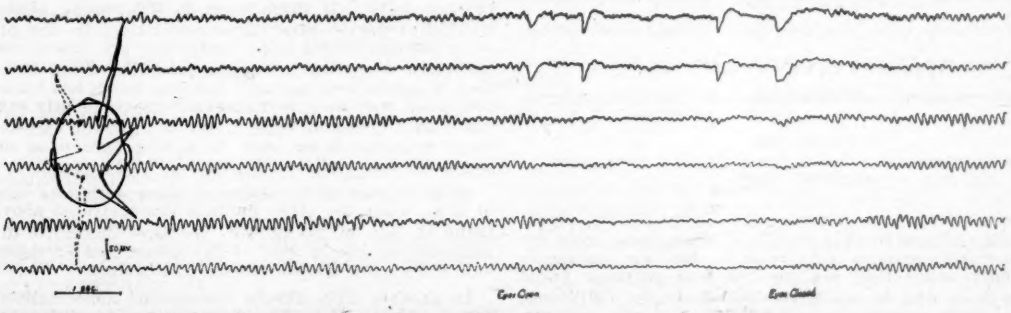

FIGURE V.
Section of a normal record. Note the blocking effect of visual stimulation, and the artefacts due to blinking seen in the channels recording from the frontal regions.

related to their site of origin in the brain. The usage of the nomenclature for the three types is still valid and useful, but it seems likely that the difference, for example, between the seizure patterns of pure *petit mal* and minor *psychomotor attacks depends on both the origin of the*

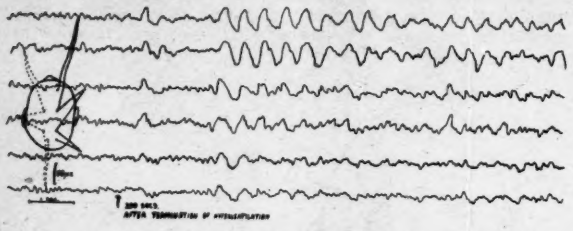

FIGURE VI.

Outbursts of sinusoidal δ activity in the post-over-breathing record of a "normal" medical student, aged twenty-six years.

disturbance and the pathways, or "circuits", involved in the maintenance of the fit. On this hypothesis it may be that *the clinical attacks of pure juvenile petit mal* and the *petit mal* in those who also suffer *grand mal* are similar resultants from different origins.

Less often are seen psychomotor seizures (10 cases), or prolonged outbursts with a similar wave pattern (15 cases). The example *illustrated (Figure X) is from the record of* a young woman who, after hyperventilation, became unresponsive and began to move as if to pull off the electrodes. When restrained from this, she *turned her attention* to a nearby light switch which she turned on and off. *She continued such* automatic activity and remained unresponsive for the duration of the abnormal record, which was composed of irregular high voltage waves of variable *frequencies down to two cycles per second,* arising from all areas of the brain. Short bursts of these irregularities continued to appear in the record for some minutes after the end of the clinical seizure, *thus confirming their true* significance (Figure XI).

These seizures have their origins in the temporal lobes and are often associated with temporal lobe abnormalities in the interseizure records and are not necessarily "idiopathic". *The temporal lobes are the highest association* areas. It is therefore not surprising that the sensory psychic phenomena in these fits are at the "conceptual level" and the motor manifestations are integrated, apparently purposeful, but really inappropriate automatisms. *These fits were once often mistaken for* hysteria. In their minor form they are still often referred to as *petit mal,* but the term "minor attacks" is preferable.

Rarely a major *convulsive seizure may be recorded* (Figure XII). The seizure record of such an attack is

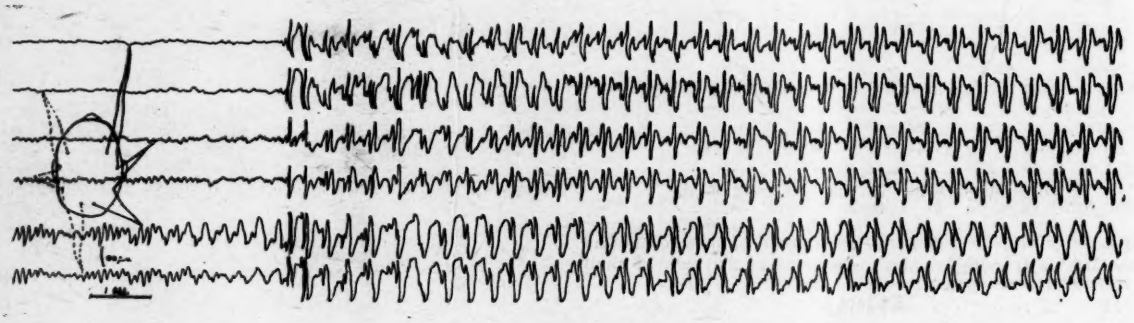

FIGURE VII.

Recording of the commencement of a clinical attack of "pure juvenile *petit mal*" in a male, aged *thirteen years. Note the* characteristic "wave and spike" pattern.

More often than a clinical seizure, a brief seizure pattern *appears in the records,* without clinical evidence of a seizure, presumably because it is too short (*Figure VIII*).

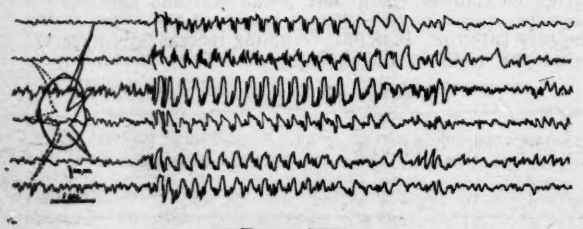

FIGURE VIII.

A "larval" *petit mal attack, recorded in a male,* aged eighteen years.

These we refer to as "larval attacks" or, if very *short,* "transient outbursts" (Figure IX). When these have the wave and spike pattern or its variants, they are diagnostic of epilepsy, since they are for practical purposes never seen in those who do not have clinical attacks (Williams, 1941a). Such patterns were seen in 69 of our patients. These patterns have the additional significance of being almost certainly idiopathic.

grossly distorted by muscle spikes and movement artefact, and the pure cortical element in the tracing can barely be *distinguished. The essential abnormal component* is the multiple spike activity.

In three other cases myoclonic jerks were observed associated with a clear-cut single spike formation (Figure XIII).

Common findings in the resting record of patients complaining of fits are paroxysmal outbursts of δ activity (*Figure XIV*). If these occur in the resting phase, or a significant period after hyperventilation, in the presence of an adequate blood sugar level, they are almost certainly *diagnostic of epilepsy in such patients.* They are rarely seen in people who have not had fits; but it has been shown that when they are, their owners very frequently suffer fits under such stress as head injury or alcoholic excess. They were virtually never seen in a large group of effective *Royal Air Force pilots* (Williams, 1944b).

Of our group of 524 patients, these outbursts were seen in 15%, while the other findings demonstrated above were found in 23% of the group. We have therefore, thus far, epileptic records in 38% of the group, and of these about *half (18%) are almost certainly idiopathic.*

In another 23% diffuse non-specific abnormalities were *found (Figure XV).* The abnormalities are diffuse, random or continuous activity with frequencies slower or faster than the normal range, and cannot be distinguished from

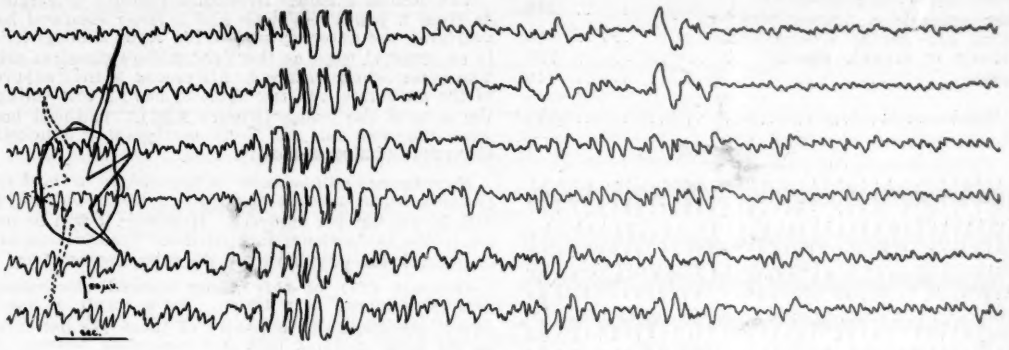

FIGURE IX.

A "transient outburst" of spike and wave activity recorded in a male, aged fourteen years.

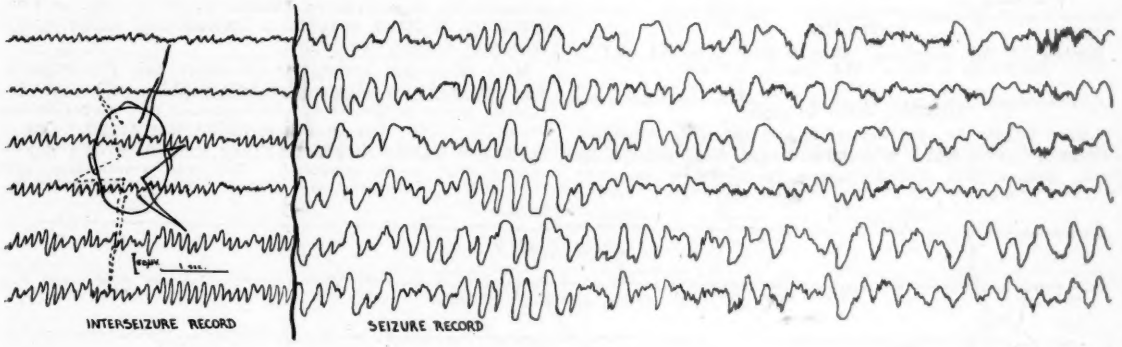

FIGURE X.

Sections from the record of a female, aged fourteen years, showing alteration in the recording during a clinical attack of psychomotor epilepsy.

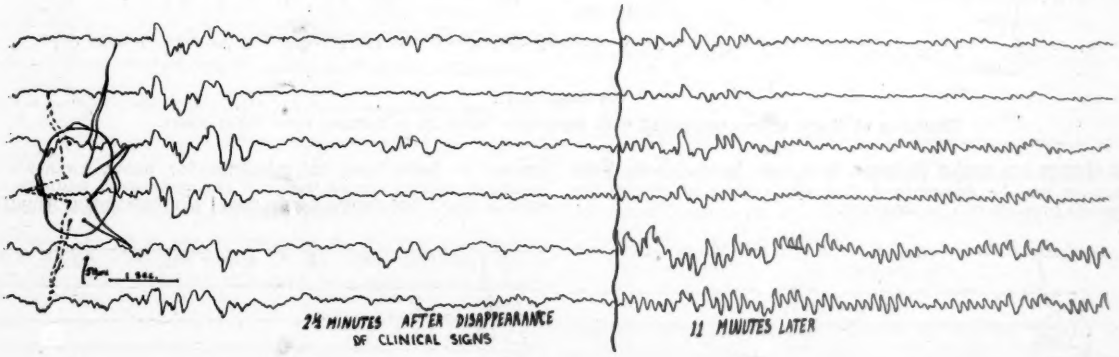

FIGURE XI.

Further sections from the record as in Figure X, showing persistence of the abnormality in the recording after the disappearance of all clinical signs of the psychomotor attack.

those sometimes seen in the records of otherwise normal people or of clinically abnormal people without epilepsy.

In another 15% the electroencephalogram gave no evidence of epilepsy, but some evidence of organic lesion (Figure XVI). (The follow-up and analysis of these records are not yet complete.)

In 127 cases (24%) the records were considered to contain no significant abnormality. *This is most frequently* so with patients who have *grand mal* only and no lesion—it is rarely so in patients who have *petit mal* alone. *Abnormal findings decrease significantly* with age. In this normal group are three subjects who "had a turn" during the recording without the production of any change in the record, so that epilepsy was *excluded entirely as a diagnosis* (Figure XVII). It may be emphasized here that this *is the only circumstance* in which the electroencephalogram excludes a diagnosis of epilepsy.

The summary of findings in the epileptic group is shown in the following tabulation:

Specific idiopathic patterns 18% Other epileptic outbursts 20%	38%
Diffuse non-specific abnormality	23%
Evidence of organic disease	15%
Normal	24%
Total	100%

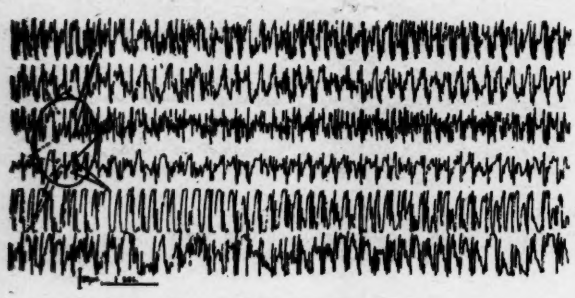

FIGURE XII.

Section of the recording taken during a *grand mal* seizure in a male, aged twenty-four years.

Organic Brain Disease.

It need hardly be said that it is the physical properties of organic disease which will influence the electroencephalogram. Site, extent, destructiveness or otherwise and rate of change are major factors. It is only in so far as these factors can be determined that the nature of the pathological process can be deduced.

The second example illustrates another principle. This is from a young boy who had a large subdural hygroma overlying the anterior half of his right hemisphere. This is an unusual case, as the child suffers classical *petit mal*. The outstanding feature in his record is the "suppression" of the outbursts, causing them to have a low voltage over the area of the lesion (Figure XIX). The cyst has since been removed, but his *petit mal* persists clinically and electroencephalographically.

No reference will be made in this section to those epileptic patients whose records suggested a lesion but whose final results are not yet analysed. However, it will be useful to study the 151 patients submitted for examination to aid in the search for possible organic brain disease.

One-half (75) of this group showed electroencephalographic evidence of a lesion. As a result of subsequent investigations, about one-third of these (28 patients) have so far been operated upon for expanding lesions. In one case no lesion was found, in 23 cases the electroencephalographic localization had been correct, and in four the lateralization but not the localization was correct. Subsequent investigations of the remaining cases revealed atrophic or degenerative lesions which do not call for operation, or failed to reveal a demonstrable lesion, and so the cases remain *sub judice*.

More important perhaps is the other half (76 subjects) who showed no electroencephalographic evidence of a lesion. Eight of these came to operation and no lesion was found in three; but in five a lesion was found. One of these five lesions was a pituitary tumour, and a normal record was to be expected. One was a subdural cyst, known to have been in existence for about twenty-five years—a normal record is one of the recognized findings in such a case. Two were parasagittal meningiomata, usually recognized as being silent in the electroencephalogram; but in reviewing our meningiomata as a group, we have noted features which we shall discuss elsewhere. The final case was one in which a deep left frontal glioma was present, and in this record we can now see features which we had previously overlooked.

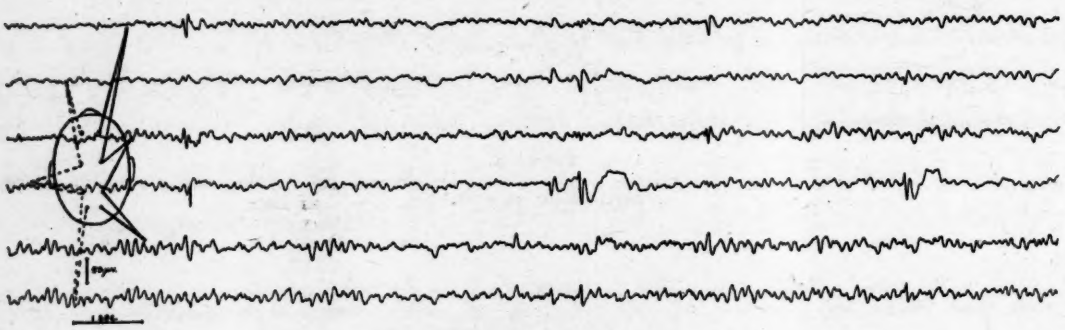

FIGURE XIII.

Examples of single spikes associated with myoclonic jerks in a female, aged eight years.

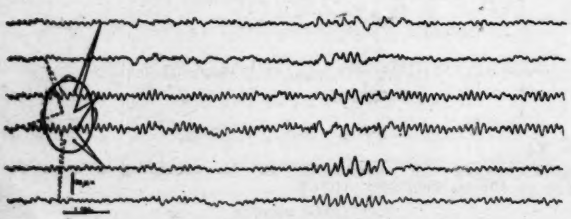

FIGURE XIV.

An outburst of δ activity seen in the resting record of a male, aged nineteen years.

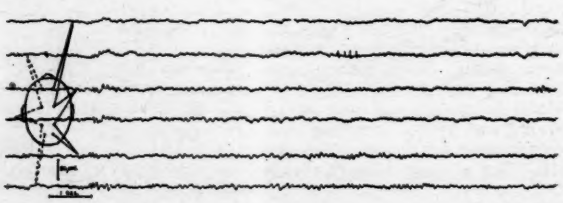

FIGURE XV.

Diffuse non-specific abnormalities in the record of a male, aged twenty-one years.

Only two examples will be given. The first is from a patient with a frontal lobe abscess (Figure XVIII). Being a rapidly destructive lesion, an abscess causes the grossest abnormality in the electroencephalogram. The example shows irregular slow δ activity arising anterior to the most frontally placed electrode on the right-hand side.

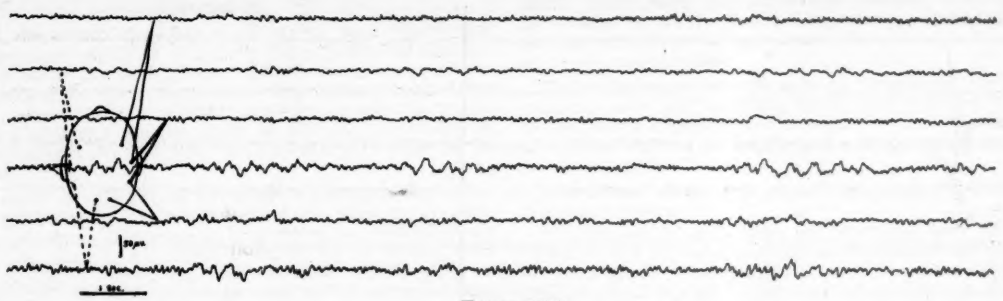

FIGURE XVI.

Delta activity in the left posterior temporal region providing evidence of an organic lesion in the record of a female, aged fifty years, complaining of epileptiform attacks.

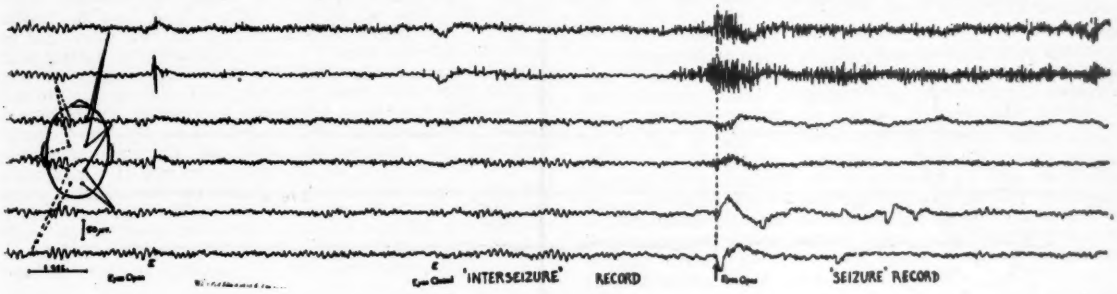

FIGURE XVII.

Sections from the record of a male, aged forty-eight years. The differences between the "seizure" and "interseizure" record are only those associated with visual stimulation and movement artefact. (The fast activity recorded from the anterior leads consists of muscle "spikes".)

It is realized that the number of our suspected lesions so far verified (32) is too small for the figures to be significant; but since the percentages do in fact correspond with larger published series (Cobb, 1950; Schwab, 1951; Culbreth, 1950) and may therefore be considered to reflect the average findings, they are given below: correctly localized, 23 (72%); correctly lateralized, 4 (12%); overlooked, 5 (16%).

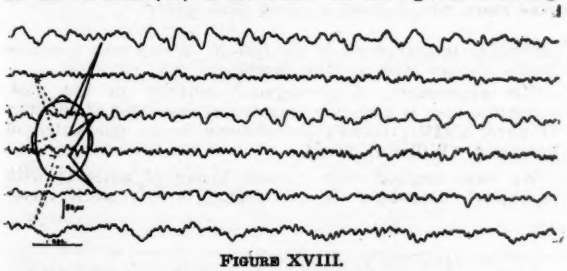

FIGURE XVIII.

Evidence of a right frontal lobe abscess in the record of a male, aged forty-one years.

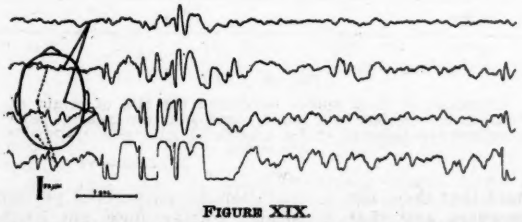

FIGURE XIX.

Suppression of abnormal activity in the area corresponding to the location of a right anterior subdural hygroma, in a male, aged three years.

Other Conditions.

We have so far either inadequate correlations with the clinical histories and autopsy results—for example, in vascular disease—or an insufficient number of cases—for example, in head injury—to make it worth while to quote figures. However, it may be useful to show some typical examples.

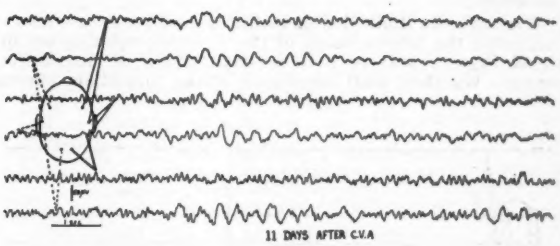

FIGURE XX.

Evidence of a destructive process in the left frontal region of a female, aged forty years, believed to have had a cerebral vascular accident.

Stated in general terms, it seems to us that a good deal of cerebral atheroma can exist without influencing the record—a fact of some value in differentiating vascular disease from tumour. However, if infarction or bleeding occurs, the record is altered in the same way as it is by other destructive lesions, such as tumours. Electroencephalographically, the two can be differentiated only by their behaviour in time, which can be observed by serial records.

We were asked to examine in the wards a hypertensive woman who had what is usually accepted as hypertensive cerebral encephalopathy. She had been unconscious, but had no focal neurological signs. The electroencephalogram showed evidence of a destructive lesion in the left frontal

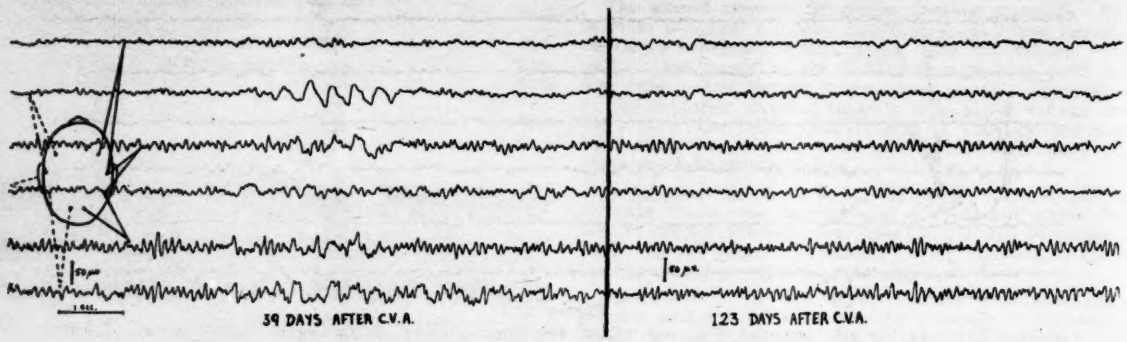

FIGURE XXI.

Disappearance of the abnormalities from the record of the female, aged forty years, as in Figure XX.

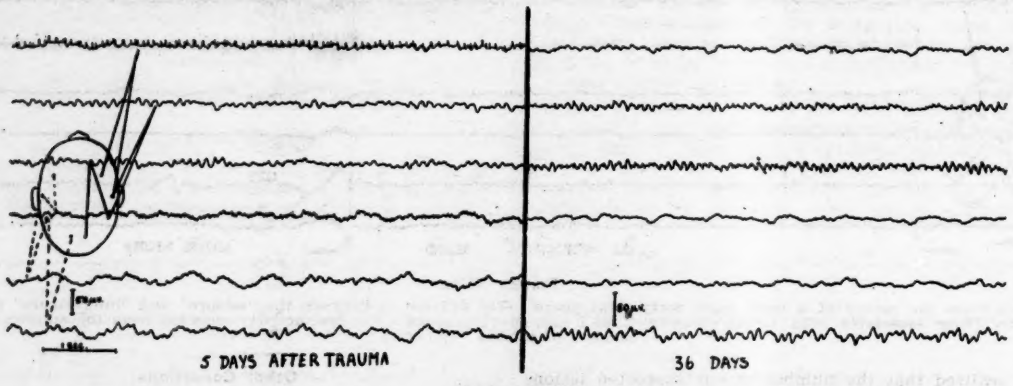

FIGURE XXII.

Sections from the record of a male, aged twenty-nine years, who suffered a closed head injury.

region (Figure XX). In four serial records over a period of three months, this record slowly returned to normal (Figure XXI). A few months later she died after a massive intracerebral hæmorrhage deep in the left hemisphere, observed at autopsy.

Through the limitation of facilities we have by no means exploited the known value of the electroencephalogram in head injury; but we have a small group of instructive cases. We shall omit discussion of the immediate effects of head injury, stating simply that the electroencephalogram can give valuable evidence of a favourable prognosis in a case of closed head injury earlier than can be determined clinically. This is shown by the reestablishment of the normal rhythms in serial records.

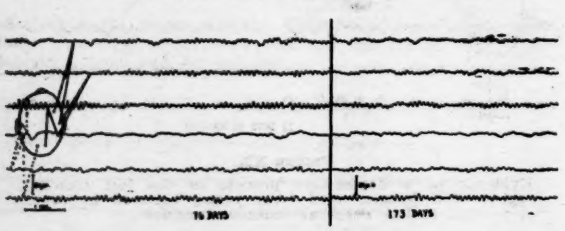

FIGURE XXIII.

Sections from later records than those in Figure XXII, showing the reestablishment of normal activities.

An example is a young man who received a closed head injury (Figure XXII). As a result he suffered right hemiparesis and complete aphasia, shown by angiography to be due to occlusion of his left middle cerebral artery. Rapid improvement in his record (Figure XXIII) heralded a slow but steady improvement of his speech; he can now converse lucidly though with a little hesitancy.

The appearance of paroxysmal activity in the post-traumatic case is a certain warning of the onset of epilepsy (Figure XXIV), though its absence is no guarantee of immunity (Williams, 1941*b*).

We have studied only a small group of patients with psychiatric disorders. In this field it is universally recognized that there are no characteristic patterns of psychotic disorders and that a normal tracing does not exclude organic disease. Interesting studies (Hill, 1944; Rey, Pond and Evans, 1949) are linking aggressive episodic behaviour with patterns of immaturity, especially of the temporal lobes, and the merging with epilepsy provides the modern counterpart of Gower's "borderland of epilepsy".

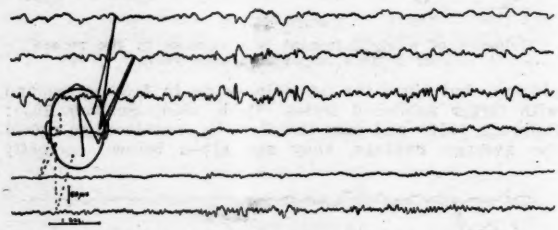

FIGURE XXIV.

Examples of focal spikes indicating the site of origin of epileptic activity in a male, aged nineteen years. The spikes are believed to be associated with post-traumatic scarring.

Conclusion.

Most laboratory methods break down just where they could be most useful in settling a difficult clinical decision between normality and disease. Electroencephalography is no better and no worse than other comparable examinations in this respect. It has probably contributed more to fundamental physiology and as much to clinical knowledge as electrocardiography had done at a comparable stage of its evolution.

The object of this survey has been to give clinicians some useful guidance in the evaluation of the electroencephalogram. We have felt pleased that when we analysed our figures, as quoted, we found them to be in accord with other published series, and so we have felt justified in quoting them as generally indicative of the present value of the electroencephalogram.

We have shown that in epilepsy the electroencephalogram rarely makes a diagnosis when an adequate clinical history fails, and that it cannot be used as a substitute for the latter. It may help a great deal in the selection of idiopathic cases and so shorten the investigation. It gives information now essential for the proper selection of drugs, and often points to the existence of an organic cause. Carefully reported and properly correlated with clinical evaluation, it should not give false positive results and so "create" epileptics. It may exclude epilepsy under certain circumstances. It has value in trauma, for prognosis especially. It has real value in the diagnosis of organic brain disease and enables us to follow its course. And yet it must be emphasized that, used alone, it is not an effective filter for epilepsy or organic disease, and its use without an adequate neurological history, examination and evaluation not only is illogical but may be misleading.

References.

COBB, W. A. (1950), "Intracranial Tumours", in "Electroencephalography", edited by Hill and Parr, London, 300.

COLBRETH, G. C. (1950), "Electroencephalography in Brain Tumour Suspects", *E.E.G. Clin. Neurophysiol.*, 2: 115.

HILL, D. (1944), "Cerebral Dysrhythmia: Its Significance in Aggressive Behaviour", *Proc. Roy. Soc. Med.*, 37: 319.

LENNOX, W. G., GIBBS, E. L., and GIBBS, F. A. (1940), "Inheritance of Cerebral Dysrhythmia and Epilepsy", *Arch. Neurol. & Psychiat.*, 44: 1161.

RBY, J. H., POND, D. A., and EVANS, C. C. (1949), "Clinical and Electroencephalographic Studies of Temporal Lobe Function", *Proc. Roy. Soc. Med.*, 42: 893.

SCHWAB, R. S. (1951), "Electroencephalography in Clinical Practice", Saunders, Philadelphia, 118.

WALTER, W. G. (1950), "Technique—Interpretation", in "Electroencephalography", edited by Hill and Parr, 66.

WILLIAMS, D. (1941*a*), "The Significance of an Abnormal Electroencephalogram", *J. Neurol. Neurosurg. & Psychiat.*, 4: 267.

WILLIAMS, D. (1941*b*), "The Electroencephalogram in Chronic Post-traumatic States", *J. Neurol., Neurosurg. & Psychiat.*, 4: 145.

WILLIAMS, D. (1944*a*), "The Nature of Transient Outbursts in the Electroencephalogram of Epileptics", *Brain*, 67: 22.

WILLIAMS, D. (1944*b*), "The Nature of Transient Outbursts in the Electroencephalogram of Epileptics", *Brain*, 67: 20, 34.

"DARAPRIM" IN THE WHAGI VALLEY OF NEW GUINEA.

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"DARAPRIM" (pyrimethamine) is known in France as malocide, the formula being 5(*p* chlorophenyl)2:4-diamino-6-ethylpyrimidine.

The idea that diaminopyrimidines might have antimalarial activity had its origin in the fertile brain of Dr. George Hitchings, who works in the Wellcome Research Laboratories, Tuckahoe, New York. Experiments on infected laboratory animals, which were carried out both in America and in London, at once confirmed this shrewd deduction. Dr. Hitchings and his team then proceeded to synthesize a comprehensive series of compounds of this kind. These were sent across the Atlantic to be tested by Dr. Rollo in the Wellcome Laboratories, London, against chicken and mouse malaria, and "Daraprim" is the product of these investigations.

These field notes on the uses of "Daraprim" as an antimalarial have been compiled during the past twelve months at the Government Native Hospital at the eastern end of the Whagi Valley, where there is a large native population of some 24,000.

Thin blood films only have been used to identify the malaria parasites.

The Whagi Valley is large and beautiful, with an equable climate; my thermometer has never registered above 88° or below 51° F. The rainfall is about 80 inches *per annum*; the elevation of the valley above sea level is 5000 feet. Mountains flanking the valley tower to 12,000 and 13,000 feet.

The natives are primitive and have not been in contact with European medicine for very long. Many have yet to learn the benefits to be derived from reporting sickness in its early stages.

Topographical features and climatic conditions are important factors in the distribution of malaria in the valley and foothills. Some areas are free, others highly malarious. The immune state of the natives is a variable factor.

A total of 306 patients suffering from malaria have received "Daraprim" treatment; in all cases positive findings were obtained on examination of thin blood films. The parasites were distributed as follows: malignant tertian parasites, 149 cases; benign tertian parasites, 100 cases; quartan parasites, 42 cases; malignant tertian and benign tertian parasites, 11 cases; benign tertian and quartan parasites, 4 cases.

Malignant tertian parasites usually disappear from thin blood films in twenty-four to forty-eight hours, benign tertian parasites in four or five days. The quartan cases were difficult, as there were so few parasites to discover in a thin film.

There was one fatal case and one patient who failed to respond satisfactorily to "Daraprim" treatment. In both cases malignant tertian infections were present.

The fatal case was that of a married man, aged forty-nine years, who lived high up in the "Bush True" and I should imagine was a "non-immune". He went on a trading trip to the "nether regions" after red bird of paradise plumes, and on his return home fell ill. After a fortnight his wife became anxious about his state of health, and informed a native medical assistant that her husband was "nigh unto death". The assistant brought the patient into hospital, where he died eight hours after his admission, in spite of 75 milligrammes of "Daraprim". He was cold, collapsed and intensely jaundiced, and neither liver nor spleen edge was palpable. He was mentally deranged and disorientated, but had no vomiting or diarrhœa; no urine examination was carried out. His hæmoglobin value was 40%, and examination of a thin blood film revealed a heavy malignant tertian infection, there being 130 parasitized red cells to 1000 red blood corpuscles. Many red cells had two, even three, ring form parasites. Numerous crescent bodies were seen.

The patient who did not respond satisfactorily to "Daraprim" treatment was a boy, aged twelve years, suffering from thyrotoxicosis complicated by malignant tertian malaria. Scanty ring forms were present in his thin blood film. He received 50 milligrammes of "Daraprim" on two days, and his clinical condition improved. Parasites were not found after twenty-four hours, but reappeared again after forty-eight hours. He was then given "Nivaquin" and appeared well.

Age Incidence.

The ages of the patients were as follows: one to six months, 13; six months to six years, 122; over six years, 171; the total, of course, being 306.

Treatment.

The objective has been not only to control the overt attack, but to prevent relapses or reinfection.

One hundred and thirty-five of the patients were aged under six years. Many had been ill for weeks or months

before their parents sought treatment for them. They were desperately ill, with profound anæmia and large spleens and livers. These and other patients received prophylactic treatment with one tablet per week as far as possible, and were followed through until their hæmoglobin value had reached 80% on the Tallqvist scale, and their livers and spleens were no longer palpable.

The fact that hepatomegaly in these children readily responded to antimalarial treatment points to the fact that the large livers in these malarious children are the result of malarial infection. After six weekly doses of 25 milligrammes of "Daraprim" neither spleen nor liver edge could be palpated in many cases.

Schedule of Dosage.

The dosage has been as follows. Children aged up to six months are given one tablet of 25 milligrammes, the dose being usually repeated the second day. The same dosage is used for children aged between six months and six years. At ages over six years the dosage is two tablets for one or two days. All patients, babies and adults alike, received weekly one tablet of 25 milligrammes as a prophylactic measure. In the case of the small children, aged up to three or four years, the tablet was disintegrated in a teaspoonful of water and swallowed "willy-nilly" with some persuasion.

Conclusion.

It has not always been possible to follow up all the patients that one would have liked to, but those who have been observed have given the greatest satisfaction. It has been possible to watch them develop from miserable, puny, anæmic children devoid of any interest in life, to children full of the joy of living.

Acknowledgement.

I have to thank Dr. Alec May, Acting Director of Public Health, for permission to publish these notes.

REGURGITATION AND VOMITING DURING INDUCTION OF ANÆSTHESIA.

By PHILIP WOLFERS, D.A.,
Melbourne.

THERE is a large class of patients, mainly found amongst surgical "emergencies", in whom the occurrence of regurgitation or vomiting during induction of anæsthesia is to be feared. The hazards attendant on these accidents and the recognition of the patients at risk are discussed at length by Morton and Wylie (1951); the points from their paper to which I should like to draw special attention are the following: (i) Regurgitation appears to be purely a passive phenomenon and is seen very frequently when anæsthesia is induced with thiopentone in a patient with a full stomach. (ii) Should regurgitation occur, it is likely to do so before even the most dextrous of anæsthetists can pass a Magill tube and inflate the cuff. (iii) In the majority of cases, the stomach can be emptied efficiently by thorough washing through a Ryle's tube. In the remainder a tube of larger bore is necessary. Nicholson (1949) points out that when blood clots are present in the stomach, it is impossible to remove them via the œsophagus.

In dealing with such patients, several techniques have been described which may be adopted to minimize the risk. With all these techniques the stomach must be emptied, as far as possible, and the Ryle's or other tube must remain *in situ* throughout the operation. If a Magill tube is used, it should be a cuffed one, as a pharyngeal pack may be dislodged if fluid rises up the œsophagus.

1. If the patient is to retain consciousness throughout the operation, local (regional) analgesia, or spinal (single or fractional dose) or epidural (lumbar or caudal, single or fractional dose) analgesia may be used.

2. If the patient is to be unconscious during the operation, one may use (*a*) simple induction of anæsthesia and intubation, (*b*) induction of anæsthesia and intubation with the patient in the sitting position, (*c*) tracheal intubation under topical analgesia before induction of anæsthesia.

The choice of a technique is governed by the experience of the anæsthetist, by the condition of the patient, by the surgeon's wishes, and by the patient's wishes. Clearly, for example, the anæsthetist whose experience is confined to the use of nitrous oxide, oxygen and ether would be foolish to resort to spinal analgesia when confronted with an operation for acute intestinal obstruction; likewise, if the surgeon demands that his patient be conscious during her Cæsarean section, one should think carefully before denying his wish.

Operation on the Conscious Patient.

Regional analgesia can be very satisfactory for both surgeon and patient if the abdomen is not grossly distended. The writer considers it the technique of choice for emergency gastrectomy in the presence of bleeding; however, the patient does occasionally suffer considerable discomfort. For this method to be successful, a gentle surgical technique is essential. The recommended steps are as follows: (i) subcostal field block and infiltration of the proposed incision; (ii) posterior (or anterior) cœliac plexus block; and (iii) paraœsophageal vagus block by the surgeon. The use of mephentermine sulphate (hyaluronidase) in the solution for cœliac block is unwise, as the solution tends to spread widely and an undesired hypotension of 60 millimetres of mercury, systolic, or even less, may result. In a personal series of 21 gastrectomies in which this technique was used, the results were uniformly satisfactory. Regional analgesia (paravertebral or field block) has also been found useful in the writer's hands in cases of early strangulated hernia.

Spinal and epidural analgesia may be considered together, as their clinical effects are similar. Spinal analgesia will certainly produce excellent relaxation, but the level of intercostal paralysis is difficult to predict unless the fractional technique, not a popular one in England or Australia, is used. The fall of blood pressure which occurs with both methods is undesirable in these patients, many of whom are already suffering from "shock". Belinkoff and Hall (1951), reporting on 180 cases of acute intestinal obstruction, state that they found "continuous spinal" analgesia the most effective anæsthetic technique. The writer's experience of spinal analgesia in this class of patient is confined to the use of low spinal block together with very light general anæsthesia for emergency prostatectomy, and in these circumstances it gave complete satisfaction. If spinal or epidural analgesia is chosen, the blood pressure should be checked frequently and if necessary supported, especially in "shocked" patients. Oxygen should be administered intranasally, and the anæsthetist should be prepared to deal with vomiting.

Operation on the Unconscious Patient.

Simple induction of general anæsthesia and immediate intubation of the trachea is a technique which has, in the writer's opinion, given rise in so many cases to inhalation of foreign material that it should be avoided when the danger of regurgitation exists. An empty stomach is not a sufficient safeguard if it is going to fill up from below as soon as unconsciousness occurs. This opinion is supported by that of Belinkoff and Hall (1951). If it is decided to conduct the anæsthesia in this way, there are several points which should receive attention.

The stomach should be washed out, not only before the patient leaves the ward, but again immediately prior to induction of anæsthesia. The Ryle's or, preferably, larger tube should not be removed from the stomach.

A sucker, turned on and functioning, should lie close to hand (for example, under the patient's pillow). A head-down tilt of up to 20°, provided that the patient can tolerate it, will aid in allowing any regurgitated material to flow out through the mouth.

Nitrous oxide, oxygen and ether or cyclopropane are safer agents for induction of anæsthesia than the thiopentone-relaxant sequence, as regurgitation in the

earliest stages is less likely to occur, and vomiting, if it occurs, will probably do so before the cough reflex is lost.

The patient may be placed in the sitting position during induction of anæsthesia with any agent, and regurgitation would then have to take place uphill. This procedure does seem to make regurgitation less likely; but its application seems to the writer to be limited by its adverse effect on the mechanisms of circulatory compensation. In a series of some 20 cases, in which anæsthesia was induced with cyclopropane, Green (1953) found it effective.

Intubation of the trachea under topical analgesia followed by the induction of general anæsthesia is the writer's method of choice in these cases, particularly for patients with acute intestinal obstruction or perforated peptic ulcer. The technique has been recommended by Nicholson (1949) for bleeding peptic ulcer, and by Belinkoff and Hall (1951) for intestinal obstruction. Dundee (1950) regards it as safe but time-consuming and distressing for the patient. That other workers have not found it unduly distressing is testified by the papers of Harang (1945), of Sadove and Gans (1950) and of Emma and Hershey (1949), who use the technique as a routine for, respectively, plastic procedures round the head and neck, major oral surgery and laminectomies.

The technique used has been as follows.

The patient comes to the anæsthetic room sucking a 25 milligramme amethocaine lozenge (recently a lignocaine lozenge), and premedicated with atropine (1/150 to 1/75 grain) and no opiate, or up to 1/6 grain of morphine. His stomach has been washed out and the Ryle's tube is in position. The lozenge is removed, 20 to 40 milligrammes of pethidine hydrochloride are slowly injected intravenously in 1% solution (Ruben and Andreassen, 1951; Wolfers, 1953), and the stomach is again emptied. After three to five minutes the mouth, pharynx, larynx and trachea are sprayed with 10% cocaine hydrochloride or 4% lignocaine solution, a Macintosh oral spray and a Macintosh laryngoscope being used. When analgesia is adequate, a lubricated cuffed Magill tube of the largest size which will fit comfortably through the glottis is introduced by direct vision and the cuff is inflated. Unconsciousness is produced at once with 2·5% thiopentone solution and anæsthesia is continued as desired.

It was found in almost all cases in the writer's series that the patient had no memory for the events immediately preceding sleep.

The writer's series of 22 cases in which this technique was used comprised the following conditions: acute intestinal obstruction, six cases (two during convalescence from an abdominal operation); perforated duodenal ulcer, four cases; appendicitis with general peritonitis, four cases; strangulated hernia (late), three cases; gastrectomy for hæmatemesis, three cases; general peritonitis following gastrectomy, one case; dissecting aneurysm, one case. Of these 22 patients, 13 were in distinctly poor condition. Anæsthesia was maintained with nitrous oxide, oxygen and pethidine in all cases; relaxation was obtained with gallamine triethiodide ("Flaxedil") in 14 and with the continuous administration of suxamethonium chloride ("Scoline") in the remaining eight. None of these patients gave rise to anxiety during the operative procedure, which in the case of one man weighing 17 stone lasted over four hours, and all made good recoveries. Of these patients, three regurgitated a considerable volume of material after the thiopentone injection.

Summary.

Attention is drawn to the danger of inhalation of regurgitated or vomited material during the induction of anæsthesia in certain cases.

The methods of anæsthesia which may be employed in such cases are reviewed and certain precautions are suggested.

A series of 22 cases is presented in which tracheal intubation was performed on the conscious patient in order to prevent the inhalation of regurgitated material.

References.

BELINKOFF, S., and HALL, O. W., JUNIOR (1951), "Anesthesia for Intestinal Obstruction", *Anesth. & Analg.*, 30: 96.

DUNDEE, J. W. (1950), "Anæsthesia for Acute Intestinal Obstruction", *Brit. J. Anæsth.*, 22: 131.

EMMA, E., and HERSHEY, S. G. (1949), "The Management of Anæsthesia for Laminectomy", *Anesth. & Analg.*, 28: 50.

GREEN, R. A. (1953), personal communication.

HARANG, H. L. (1945), "Anæsthesia for Head and Neck Surgery with the High-frequency Electrosurgical Unit", *Anesth. & Analg.*, 24: 119.

MORTON, H. J. V., and WYLIE, W. J. (1951), "Anæsthetic Deaths due to Regurgitation or Vomiting", *Anæsthesia*, 6: 190.

NICHOLSON, M. J. (1949), "Anesthesia for the Poor Risk", *Anesth. & Analg.*, 28: 98.

RUBEN, H., and ANDREASSEN, A. K. (1951), "Pharmacological Effects of Pethidine on the Larynx as seen during Intubation", *Brit. J. Anæsth.*, 23: 33.

SADOVE, M. S., and GANS, B. J. (1950), "Anesthesia in Hospital Oral Surgery", *Anesth. & Analg.*, 29: 288.

WILSON, D. S. (1952), "Anæsthesia for the Aged", *Anesth. & Analg.*, 31: 343.

WOLFERS, P. B. (1953), "Intravenous Pethidine in Anæsthesia", *Brit. J. Anæsth.*, 25: 244.

THE EFFECT OF ISONIAZID ON GUINEA-PIGS PREVIOUSLY VACCINATED WITH B.C.G. AND INFECTED WITH STRAINS OF MYCOBACTERIUM TUBERCULOSIS ALREADY SHOWING VARYING RESISTANCE TO THIS DRUG.

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SINCE isoniazid and its derivatives were introduced as antituberculosis chemotherapeutic agents, numerous papers have appeared in the medical Press, dealing with their effectiveness, their chemical and pharmaceutical properties and toxic effects *et cetera*. Many publications have dealt with the development of resistance in the *Mycobacterium tuberculosis* against this new drug (Medical Research Council, 1952; Incze, 1953*a*; Fisher, 1952). It was stated that, under certain circumstances, the acquired resistance of *M. tuberculosis* might revert to sensitivity or the resistance level might be reduced considerably. It is not yet clear whether the presence of streptomycin and PAS is essential to produce this reversion, or whether it may occur spontaneously in their absence (Incze, 1953*b*). Concerning the importance of developed resistance, it has been shown that there is a resistance level beyond which further treatment does not greatly benefit artificially infected laboratory animals. This resistance level is around one microgramme per cubic centimetre, bacilli being still able to grow in the presence of one microgramme per cubic centimetre of isoniazid in culture media. It was shown by the present writer that guinea-pigs infected with strains with higher resistance than one microgramme of isoniazid per cubic centimetre did not respond markedly to the isoniazid treatment used. These experiments were carried out on guinea-pigs with no inborn or acquired resistance (Incze, 1953*c*).

Though it is a well known fact that the guinea-pig has a very low inborn resistance and that a "Mantoux-negative" guinea-pig has no acquired resistance, we do know that, by B.C.G. vaccination, a certain degree of protection can be achieved (Rich, 1951; Dubos *et alii*, 1953). We used for this experiment B.C.G. vaccinated animals, supposing that the pathological process would imitate more closely the adult type of tuberculosis, and that the conclusions drawn would be more applicable to clinical human tuberculosis than as if completely unprotected animals were used.

Experimental Methods.

Twelve male "Mantoux-negative" guinea-pigs of approximately equal weight (500 grammes) and age (four months) were employed for this experiment, and were maintained under similar living conditions. We vaccinated them with

0·1 cubic centimetre of an emulsion of B.C.G. containing 0·05 milligramme of bacilli (Commonwealth Serum Laboratories), and, from the date of vaccination, every fourteen days we tested the cutaneous tuberculin reaction. In six weeks all animals gave positive skin reactions in response to the Mantoux test, an intracutaneous injection of 0·1 cubic centimetre of first strength PPD (0·00002 milligramme) being given, and, if the result was negative, we repeated the test with 0·1 cubic centimetre of second strength PPD (0·005 milligramme). (See Table I.)

After conversion of the skin reaction, we separated the guinea-pigs into three groups of four individuals and infected them with virulent tubercle bacilli, using an emulsion of 0·2 milligramme of bacilli divided into halves; one half was given subcutaneously in the left leg, the other was given as an intratracheal spray. The first, or test group, received bacilli completely sensitive to isoniazid. The second group was infected with tubercle bacilli resistant to isoniazid in a concentration of one microgramme per cubic centimetre; the third group received the same amount of highly resistant strains (200 microgrammes per cubic centimetre).

After a fortnight we sacrificed one animal from each group (Figure I, numbers 1, 5 and 9), to see if the infection was progressing, but the post-mortem examinations gave no positive results, no macroscopic organic lesions being detected. For this reason we did not immediately start the treatment of the animals, but we waited for two months, examining the animals clinically every week. When clinical signs appeared, consisting of inguinal lymph gland enlargement on the eighty-second day after infection, we started the isoniazid treatment with a daily dose of 7·5 milligrammes per kilogram of body weight. We administered the isoniazid orally, carefully mixing the powdered isoniazid with the dry food of the animals. During the treatment we did not notice any significant change in the health of the animals, except that they all gained some weight. We continued treatment until the end of the sixth week, when we sacrificed our animals and very careful post-mortem examinations were carried out. Before killing the animals, we examined them clinically and noticed that the enlarged lymph glands of the groin on the side corresponding to the injections disappeared in the group infected with isoniazid-sensitive organisms, were reduced in size in the group infected with strains resistant to one microgramme of isoniazid per cubic centimetre, but were considerably enlarged in the group infected with strains of high resistance (200 microgrammes per cubic centimetre).

On post-mortem examinations we were not able to detect any macroscopic pathological change in the animals in the first group. In the second group a pepper-size caseous lymph gland was found in the left groin of guinea-pig number 7 and an enlarged paratracheal lymph gland was present in guinea-pig number 8, but some miliary nodules were found in the spleen of all the animals in this group. In the third group we found very advanced, generalized tuberculosis in every guinea-pig, consisting of major lymph gland involvement of the groin and of the paralumbar lymph glands. In guinea-pig number 10 caseous pneumonia and extensive paratracheal lymph gland involvement were present. In guinea-pig number 12 an enlarged caseous paratracheal lymph gland was detected. The spleens of all three animals of this group were enlarged and numerous caseous foci were found (see Figure I).

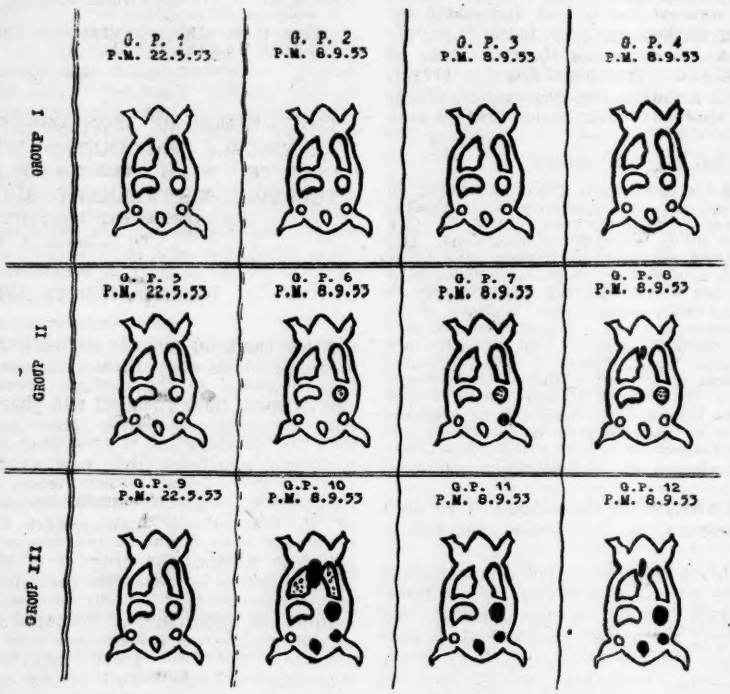
GROUP I
G. P. 1 P.M. 22.5.53
G. P. 2 P.M. 8.9.53
G. P. 3 P.M. 8.9.53
G. P. 4 P.M. 8.9.53
GROUP II
G. P. 5 P.M. 22.5.53
G. P. 6 P.M. 8.9.53
G. P. 7 P.M. 8.9.53
G. P. 8 P.M. 8.9.53
GROUP III
G.P. 9 P.M. 22.5.53
G.P. 10 P.M. 8.9.53
G.P. 11 P.M. 8.9.53
G.P. 12 P.M. 8.9.53

FIGURE I.

Discussion.

Figure I shows clearly that the treatment favourably influenced the tuberculous process in the first group of guinea-pigs infected with *M. tuberculosis* sensitive to isoniazid. In the second group the treatment was not so effective, being unable to prevent the tuberculosis from becoming generalized. However, the progress of the disease was not so malignant as it was in the third group, in which the treatment did not influence the progress of the disease at all. The impression is that the isoniazid treatment is effective in B.C.G. vaccinated guinea-pigs only if the infecting agent was originally sensitive to isoniazid. As soon as the bacilli develop a resistance against this drug which reaches the level of one microgramme per cubic centimetre, the effect of the drug is very much reduced, and it becomes completely ineffective when the bacilli are highly resistant.

What would occur if the isoniazid treatment was continued for a longer period remains an open question. This matter is under current investigation in this laboratory.

It still remains to be investigated whether the same will apply in human tuberculosis as in the B.C.G.-vaccinated experimental animals.

The main task is how to prevent the development of resistance. This has already been attempted by combining the various chemotherapeutic agents; but if, in spite of these precautions, the resistance to the drug still develops and reaches a very high level, continuance of the medication may be unwise.

Conclusions.

1. It is evident that after a period of treatment with isoniazid, isoniazid-resistant varieties of *M. tuberculosis* will develop.

2. When animals are infected with bacilli of various degrees of resistance to isoniazid and treated with the same drug, the treatment may be ineffective if the resistance level passes the drug concentration of one microgramme per cubic centimetre.

3. It would be highly advisable for pathological laboratories as a routine procedure to check the bacilli produced by patients, and if the resistance level passes one microgramme per cubic centimetre and clinically and radiologically no further improvement is evident, isoniazid therapy should be stopped and other treatment substituted.

4. There is no essential difference between the response to isoniazid of the unprotected and of the B.C.G.-vaccinated guinea-pigs infected with isoniazid-resistant varieties of *M. tuberculosis*.

TABLE I.

Mantoux Test, May 25, 1953.

Group.	Number of Guinea-pig and Degree of Reaction to P.P.D.			
Group I	1 +++	2 +++	3 ++	4 +
Group II	5 +++	6 +	7 +++	8 +++
Group III ..	9 +++	10 +++	11 +++	12 +++

Summary.

An experiment was carried out on B.C.G.-vaccinated guinea-pigs in order to determine the isoniazid resistance level of *M. tuberculosis* beyond which no further clinical improvement is to be expected. This resistance level is about one microgramme per cubic centimetre.

References.

DUBOS, R., SCHAEFER, W. B., and PIERCE, C. H. (1953), "Anti-tuberculous Immunity in Mice Vaccinated with Killed Tubercle Bacilli", *J. Exper. Med., 97: 221.*

FISHER, M. W. (1952), "The Altered Growth Characteristics of Isoniazid Resistant Tubercle Bacilli", *Am. Rev. Tubero., 66: 626.*

INCZE, J. S. (1953a), "The Development of Isoniazid Resistance in Mycobacterium Tuberculosis: Preliminary Publication", *M. J. AUSTRALIA, 1: 883.*

INCZE, J. S. (1953b), "Reversibility of Isoniazid Resistance Developed in Mycobacterium Tuberculosis", *Dis. Chest*, in the press.

INCZE, J. S. (1953c), "Effect of the Isoniazid on Guinea-Pigs Infected with Isoniazid Resistant Strains of Mycobacterium Tuberculosis", *Dis. Chest*, in the press.

MEDICAL RESEARCH COUNCIL OF THE PRIVY COUNCIL (1952), "The Treatment of Pulmonary Tuberculosis with Isoniazid: An Interim Report to the Medical Research Council by their Tuberculosis Chemotherapy Trials Committee", *Brit. M. J.*, 2: 735.

RICH, A. (1951), "Pathogenesis of Tuberculosis", 487.

Reports of Cases.

OBTURATOR HERNIA: SOME DIFFICULTIES IN PRE-OPERATIVE DIAGNOSIS ILLUSTRATED BY TWO CASES WITH UNUSUAL FEATURES.

By THOMAS F. ROSE,

From the Department of Surgery, The Royal North Shore Hospital of Sydney.

Case I.

CASE I was a case of incarcerated right obturator hernia with pain of sciatic nerve distribution. The patient, a woman, aged seventy-six years, was admitted to the Royal North Shore Hospital of Sydney with the history that during the previous two years she had suffered intermittent attacks of shooting pain, which commenced in the right buttock and radiated down the posterior aspect of the right thigh to the outer aspect of the leg and foot. These attacks would last from minutes to hours and occur at intervals of from days to weeks. They were frequently accompanied by generalized abdominal colic and vomiting, which made the sciatic pain worse. It was noticed, too, that a heavy meal would often precipitate an attack an hour or two after it was eaten. Six months after these attacks commenced, radiological studies of the gastro-intestinal tract were carried out in a pain-free interval, and a normal alimentary canal was found. Four days before her admission to hospital a very severe attack of sciatic pain commenced. This was accompanied by colicky pain localized to the lower part of the abdomen, vomiting and constipation. Two days later, abdominal distension appeared.

On her admission to hospital the patient was seen to be an elderly woman in no apparent distress and in reasonable condition. However, her tongue was furred and dry. Her temperature was normal, her pulse rate was 100 per minute, and her respirations numbered 20 per minute. The abdomen was a little distended and some visible peristalsis from left to right was present. There was some tenderness in the right iliac fossa. The percussion note was hyper-resonant all over the abdomen. Auscultation revealed a few borborygmi. The hip joints and spine were normal. There were no external hernias and no swelling or tenderness in the right femoral triangle or buttock. Rectal and pelvic examinations revealed no abnormalities save for a little tenderness high up on the right side of the pelvis. A plain radiograph of the abdomen revealed dilated loops of small bowel with at least one fluid level.

After the stomach had been emptied by means of an indwelling Ryle's tube, and intravenous therapy had been instituted to correct the dehydration, laparotomy was performed and a right obturator hernia was found. A segment of the terminal part of the ileum, one foot from the caecum, was found to be partially incarcerated in the hernia so as to constitute a Richter's hernia. Fortunately the affected bowel was still viable, though on its release it was seen that the constricted portion was tissue-paper thin. The hernia itself was left alone, as the patient's condition was deteriorating. Convalescence was uneventful, and the patient went home on the fourteenth day.

This patient has been followed up to date, two years after her operation. During this time she was unfortunate enough to sustain a pertrochanteric fracture of the right femur, which united with an excellent functional result within six months. She has also had at least three further attacks of colicky lower abdominal pain accompanied now by pain of typically obturator distribution. These attacks all occurred after heavy meals and were not accompanied by abdominal distension or constipation. In the past six months light meals only have been eaten and no further attacks have eventuated. In view of the patient's age no further surgical treatment is contemplated.

Comment.

Owing to the sciatic distribution of the pain, a confident diagnosis was made of the very uncommon sciatic hernia. However, this diagnosis was not substantiated, nor was any reason found for the peculiar pain distribution. It is interesting to note that afterwards, when the hernial symptoms were recurring, the pain was obturator in distribution.

Case II.

The second case was one of acute appendicitis in an obturator hernia. A female patient, aged sixty-six years, had been well until ten hours before her admission to hospital, when she had severe generalized abdominal colic accompanied by constant pain along the inner side of the right thigh radiating down to the knee. This pain was worse when she moved the thigh, so that she limped as she walked. The pain was eased when the thigh was held in flexion.

Examination disclosed the patient to be a sick woman, whose temperature was 100° F. Her pulse rate was 120 per minute, and her respirations numbered 25 per minute. Tenderness was present above the symphysis pubis and there was slight generalized abdominal distension. Pelvic and rectal examinations disclosed tenderness on the right side of the pelvis, where there was a suggestion of fullness but no definite mass. Pressure on this caused an exacerba-

tion of the thigh pain. The thigh was held in slight flexion and external rotation. Hip movements were normal, but the psoas and obturator signs were strongly positive. There was a little tenderness in the right femoral triangle, but no swelling.

A diagnosis of a strangulated obturator hernia was made.

At operation a right obturator hernia was found, with the distal half of an acutely inflamed appendix due to fæcolith obstruction jammed in the hernia. When the appendix was mobilized out of the hernia the tip was found to be perforated and the sac full of pus. Appendicectomy was performed. The sac was left *in situ* because of the risk of infection which its removal might entail. Convalescence was uneventful.

A follow-up examination of the patient one year later disclosed no further symptoms referable to the hernia.

Comment.

The diagnosis of an obturator hernia was correct, but its contents and their condition were not diagnosed. This is hardly to be wondered at, because the only clinical differences which this case presented from those of an obturator hernia containing incarcerated bowel was the absence of a previous history of similar attacks and the presence of a mild temperature.

Discussion.

Obturator hernias are notoriously difficult to diagnose; according to Watson (1938), only one in four is diagnosed before operation, mainly because most patients have no symptoms or signs referable to irritation of the obturator nerve, and fewer still have a swelling in the femoral triangle. Almost all such hernias are of the Richter type, so that the patients have partial intestinal obstruction, abdominal distension being an early prominent sign. In a right-sided hernia with ileal involvement large bowel obstruction is so mimicked that a plain radiograph of the abdomen may be needed to diagnose the level of obstruction. Consequently, the pre-operative diagnosis is usually that of an intestinal obstruction of obscure causation.

This is well shown by two other recent cases of obturator hernia, each patient having a strangulated Richter hernia, which occurred recently at the Royal North Shore Hospital. One patient was under the care of Dr. Eric Langley and the other had been the subject of a previous report in this journal (Rose, 1950). Both patients were diagnosed as suffering from intestinal obstruction of unknown ætiology, because neither had signs or symptoms referable to the obturator canal. On the other hand, the presence of the Howship-Romberg syndrome *plus* abdominal pain does not necessarily mean that a strangulated obturator hernia is present, as may be shown by the following two cases.

Case III.

The patient, a female, aged sixty-five years, had an acutely inflamed appendix adherent to the right side of the pelvis simulating a right-sided obturator hernia. Sixteen hours prior to her admission to hospital this patient had an attack of generalized abdominal pain which eight hours later radiated to the right iliac fossa. This pain was accompanied by pain down the inner aspect of the right thigh as far as the knee. The patient vomited several times during her attack, and this aggravated the thigh pain.

Examination of the patient on her admission to hospital disclosed her to be a sick-looking woman with a normal temperature and a pulse rate of 85 per minute; her respirations numbered 25 per minute. Her tongue was furred and dry. There was some tenderness in the right iliac fossa, but no rigidity. Though the right hip joint movements were normal, an obturator sign was present. There was no tenderness or swelling in the right femoral triangle. Pelvic and rectal examinations disclosed a tender, thick area on the right side of the pelvis, pressure on which accentuated the obturator pain, causing it to shoot down the thigh. The diagnosis of a strangulated right-sided obturator hernia was made.

However, operation showed that an acutely inflamed, obstructed appendix was lying just below the pelvic brim on the right side. No obturator hernia was present. Appendicectomy was performed and an uneventful convalescence followed. A follow-up examination of the patient twelve months later disclosed no further illnesses.

Case IV.

The patient, a female, aged seventy years, had a pelvic pericolic abscess due to diverticulitis of the sigmoid colon, simulating a strangulated left obturator hernia. With no history of previous illness, this patient had had left-sided lower abdominal pain for five days. It was at first accompanied by diarrhœa and later by constipation. It was associated with repeated vomiting and pain along the inner aspect of the left thigh running down to the knee.

Examination of the patient showed her to have a temperature of 101° F. and a pulse rate of 120 per minute; her respirations numbered 24 per minute. She was flushed and her tongue was furred. Tenderness and some rigidity were present on palpation in the left iliac fossa. A pelvic and rectal examination revealed tenderness and some fullness on the left side of the pelvis, pressure on which caused the obturator pain. Examination of the hip joints and spine revealed no apparent abnormalities.

At operation a left pericolic abscess (shown later to contain *Bacterium coli* only) was found attached to the left pelvic brim. It was arising from an area of diverticulitis in the sigmoid colon. As the bowel was not ruptured, simple drainage of the abscess was performed with no proximal colostomy. No obturator hernia was found. Convalescence was uneventful, the drainage ceasing in a week, when the bowel commenced to function satisfactorily.

Six months later the patient was well and had had no recurrence of her pain.

Summary.

Two further cases of obturator hernia are reported. One was interesting in that sciatic pain was present instead of obturator pain. In the second, the sac was occupied by a ruptured appendix.

Difficulties in the diagnosis of a strangulated or incarcerated obturator hernia are found because there are often no local symptoms and signs pointing to involvement of the obturator canal.

It is pointed out that the presence of abdominal pain *plus* pain of obturator distribution (Howship-Romberg syndrome) does not necessarily mean that an obturator hernia is present. Two cases are mentioned to illustrate this; in the first, an acutely inflamed appendix was lying on the right pelvic brim irritating the obturator nerve, whilst in the second a pericolic abscess mimicked a left obturator hernia.

References.

ROSE, T. F. (1950), "Obturator Hernia: A Case Report", M. J. AUSTRALIA, 2: 260.

WILSON, L. F. (1938), "Hernia", Second Edition, Kimpton, London.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"A Handbook of Operative Surgery: Surgery of the Biliary Tract, Pancreas and Spleen", by Charles B. Puestow, M.D., Ph.D. (Surg.); 1953. Chicago: The Year Book Publishers, Incorporated. 8¼" × 6", pp. 370, with 72 illustrations. Price: $9.00.

Intended as an additional guide to those who have received or are receiving competent surgical training.

"Parsons' Diseases of the Eye", by Stewart Duke-Elder, K.C.V.O., Ph.D., D.Sc., LL.D., M.D., F.R.C.S., F.A.C.S.; Twelfth Edition; 1954. London: J. and A. Churchill, Limited. 8½" × 5½", pp. 614, with 22 plates in colour and 465 text figures. Price: 42s.

This standard text-book has been completely revised and partly rewritten since the previous edition was published in 1948.

The Medical Journal of Australia

SATURDAY, JANUARY 2, 1954.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE JOURNAL AND THE BRANCHES.

THE MEDICAL JOURNAL OF AUSTRALIA is owned and controlled by the Australasian Medical Publishing Company, Limited. The Company was formed in the year 1914 by the appointment from each of the six Branches in Australia of three members, who comprised the Company. One of each of the group of three members was appointed to be a Director. Later on, for convenience in working two Directors were appointed from New South Wales because the journal was produced in Sydney. The Directors control the journal and meet twice each year to consider its progress and also the progress of the other activities of the Company. It is thus clear that, indirectly, the journal is the property of the Branches. It is distributed amongst the members of the several Branches as a result of an agreement between the Federal Council of the British Medical Association in Australia and the Company. The Directors appoint the Editor and his staff; they also appoint the Manager of the Company, who also acts as the Manager of the journal. In order that the Branches may be efficiently served, the Editor has in each State other than New South Wales a representative who acts as a liaison officer between the Branch Council and himself. The Directors also arrange that the Editor of the journal shall from time to time visit the several Branches in order to make himself acquainted at first hand with their doings and learn of their difficulties. The primary object of the British Medical Association, as all its members are aware, is the promotion of the medical and allied sciences. Towards this end, each of the Branches holds from time to time scientific meetings at which papers are read and discussions are held. The Branches also hold clinical meetings for the demonstration of cases and members are given an opportunity of asking questions and improving their knowledge. These papers and discussions are given to the representative of the Editor, who sends them on for publication. We may thus conclude that every Branch has an equal stake in the journal and that the members, whether they live near to the source of its production or far away, are or should be equally concerned in its welfare.

A few weeks ago, a prominent member of the Victorian Branch, in a discussion on the journal, remarked that he felt that THE MEDICAL JOURNAL OF AUSTRALIA was "chiefly Sydney". This statement was stoutly denied, and as a result of it a survey was made of all the signed contributions to the journal over a period of twelve months. The result of that survey is set out in the accompanying table.

TABLE I.
Year Ending June 27, 1953.

Branch.	Members.	Original Articles.	Reports of Cases.	Clinical Meetings.	Correspondence.
New South Wales	3574	82	33	7	60
Victoria ..	2377	85	9	11	31
Queensland ..	1035	27	5	—	15
South Australia	733	15	5	7	5
Western Australia ..	514	15	3	—	8
Tasmania ..	208	7	—	—	3
Australian Capital Territory ..	—	1	—	—	1
Overseas ..	—	7	2	—	9

It will be noted that included in this table are the numbers of members of the several Branches. It is clear that the contributions to the journal cannot be compared unless consideration is given to the number of Branch members. The figures quoted are taken from the latest Branch reports. Since these reports were issued, in several instances great increases in membership have taken place. From the table it will be clear that Victoria makes a very good showing. It leads New South Wales in the number of original articles and in the number of reports of clinical meetings, even without any consideration of the numbers of Branch members. Considered from the proportional membership point of view, the Western Australian Branch appears to have contributed more original articles than any other. Whether a statistician would come to any other conclusion than this we do not know. Of some importance is the fact that Queensland, Western Australia and Tasmania have not forwarded any reports of clinical meetings. This is to be regretted. A great deal depends on the way in which clinical meetings are held. In New South Wales, when a clinical meeting is held at a hospital, a member of the hospital staff demonstrating a patient has a room where he waits with his patient for visitors to appear, to whom he can demonstrate the interesting features in the patient's history and present the patient for examination by the visitor. This is probably the most valuable method of holding a clinical meeting from the point of view of the actual visitor to the meeting. From the publication point of view it is not the best method because none of the questions asked or the explanations given are recorded. From the point of view of the reader of the journal who cannot attend a clinical meeting, the

best method is that reported by the Melbourne Pædiatric Society where patients are shown, histories are read, and an open discussion takes place and is reported. On this account the Editor of the journal has for many years regarded the reports of the Melbourne Pædiatric Society as the most valuable clinical reports which he receives. When the method pursued in New South Wales is followed, the bare clinical reports are sent to the journal office and the journal staff has to make up an account for publication from the notes sent. On rare occasions, when what we may call the "isolationist" method of demonstration has been used, members have, on request, sent specially worded reports. But even these are not accompanied by any record of discussion. We must not lose sight of the fact that some of the hospitals in Australia have their own "Clinical Reports" in which interesting cases are recorded. This fact may account for the smallness of the number of reports of cases received from certain of the Branches. The correspondence columns are open to every member who cares to send a contribution, and contributions are accepted provided they are couched in acceptable terms.

The result of the survey of contributions is published in order to reawaken interest where it needs to be stimulated. The journal exists so that it may be of use to members of the Branches, but sight should never be lost of the fact that it is the members themselves who have to create it.

Current Comment.

NIPPLE DISCHARGE, CANCER AND PAPILLARY DISEASE OF THE BREAST.

IF a patient with a discharge from the nipple has a palpable mass in the breast, the discharge becomes of secondary importance. The mass is explored as any lump in the breast would be. If, however, there is discharge from the nipple without a palpable tumour, long-standing and difficult clinical problems arise. In particular, two questions ask for an answer. First, is it possible that there is a cancer too small or too poorly defined to be felt? Second, if not cancer, is there a lesion present which indicates increased risk of development of cancer? Since the most common benign lesion responsible for nipple discharge is papillary disease, we must then ask what is the cancer-producing potential of papillary disease or of the breast tissue which has developed it. In raising these questions, A. R. Kilgore, R. Fleming and M. M. Ramos[1] point out that a wide range of views has been published in the attempts to answer them. They reconsider the matter in the light of their own experience and their reading of the literature, and rephrase the problems in two further questions. First, what is the likelihood of cancer being present when there is nipple discharge with no mass palpable? Second, does or does not a woman with papillary disease run more risk of developing cancer than the average woman? Statistics are presented, various problems are discussed, and a series of conclusions are drawn which merit thought. The first conclusion is that a secretory discharge which is either grossly milky or is found to contain secretory elements when examined microscopically appears to be of little significance and to require no more than observation for a change of character. On the other hand, a serous discharge without secretory elements will, in one-third or more of the cases, be found at some time to contain blood; such a discharge, therefore, carries significance potentially similar to that of one containing blood. In the presence of blood-containing discharge without a palpable lump, the chance that cancer is present is about one in twenty to one in twenty-five; for this reason alone the source of bleeding should be found and its nature determined. If cancer is not present, probably bleeding is coming from papillary disease. There is wide and apparently irreconcilable disagreement about the relationship of papillary disease to the development of cancer; the conviction of Kilgore, Fleming and Ramos is that its presence indicates risk appreciably above the average. Of the methods of locating and removing a single lesion of papillary disease in the absence of a palpable lump, none is uniformly successful; and if such a lesion is found, the chance of leaving other undiscovered lesions elsewhere in the breast is considerable. For these reasons, it is advised that the woman beyond the age of forty years who has a blood-containing discharge from the nipple, or who has demonstrated papillary disease, should consider the acceptance of mastectomy, extended to radical operation if cancer is discovered. Even younger women with multiple lesions are offered similar advice.

[1] *Surg., Gynec. & Obst.*, June, 1953.

CORONARY HEART DISEASE AND PHYSICAL ACTIVITY OF WORK.

THE possible relationship of physical activity to coronary heart disease has been discussed a great deal, especially in relation to compensation claims. The tacit assumption of most discussions of this kind is that physical activity either has no effect, or causes, aggravates or precipitates coronary heart disease. It cannot be said that the issue has been settled in any scientific sense, even though court decisions, particularly in relation to the precipitation of acute and fatal attacks, have been given. However, the line of thought is taken in quite another direction by a recent investigation by J. N. Morris, J. A. Heady, P. A. B. Raffle, C. G. Roberts and J. W. Parks.[1] Their report is one of a series on the epidemiology of coronary disease. The study began with observation of transport workers and the finding that one group, the conductors, whose job obviously involved considerable physical activity, had a different experience of coronary heart disease from the drivers, whose job obviously involved less physical activity. The conductors had a low incidence of coronary heart disease and, more important, the disease they had was less severe and less often rapidly fatal. Analysis of the experience of postal workers and civil servants showed a similar trend when postmen were compared with sedentary workers. In order that the matter might be followed up the following provisional hypothesis was stated:

> Men in physically active jobs have a lower incidence of coronary heart disease in middle age than men in physically inactive jobs. More important, the disease is not so severe in physically active workers, tending to present first in them as *angina pectoris* and other relatively benign forms, and to have smaller early case-fatality and a lower early mortality-rate.

The hypothesis was tested in three ways. First, the evidence was enlarged by further observation of transport workers over a subsequent year. The early mortality rate of conductors and postmen was again found to be lower than that of the drivers and telephonists. There is reason to believe that this "early mortality" (that is, death in the first weeks of the first clinical attack) makes up the greater part of the total coronary mortality in middle-aged men; so, as a second test, an analysis was made of the Registrar-General's occupational mortality data for the period 1930 to 1932. This showed that at forty-five to sixty-four years of age heavy workers had half the mortality from coronary heart disease of light workers. As a third test, a sample of death certificates of patients dead of coronary heart disease in London and the Home Counties during March, 1952, was examined; this suggested that heavy workers died less often than light workers in their

[1] *Lancet*, November 21 and 28, 1953.

first clinical attack and tended more often than light workers to have the disease in chronic form.

That is as far as the investigation has gone. Morris and his colleagues point out that none of the tests which have been applied falsify the hypothesis; some support it strongly, some weakly. What they, for convenience, call "the protection against coronary heart-disease associated with physical activity in work" has been demonstrated in some measure in a number of ways in national, regional and group statistics: in the mortality of three social classes separately, and of the larger occupational groups individually; in the morbidity from coronary heart disease of several grades of workers, as well as in their mortality; over the range of middle age and within it; when the standard of diagnosis was held constant and was probably high, and when it may have varied widely; currently and twenty years ago; before the disease was commonly diagnosed and since; on two separate gradings of physical activity; on an approach to the problem through the study of "populations" as well as by the collection of cases. It is reasonable to agree that reliance may be placed on the cumulative effect of the regularities that have been demonstrated, however inadequate some individual pieces of evidence by themselves obviously are. The constancy of the association in such varying circumstances encourages the hope expressed by Morris and his colleagues that we are dealing with a causal relationship. As they state, the hypothesis may be regarded as a useful one which has so far survived.

The further implications of the hypothesis have yet to be determined. A number of relevant questions are raised in this paper, although none is answered with finality. The possible association and, perhaps, confounding of psychological with physical factors requires a good deal of thought. Another consideration is the possible effect of physical activity on coronary circulation. One relevant observation made is that men engaged in more active work appear to have not merely a lesser total incidence of coronary disease, but also, within this feature, more angina. It is suggested, in an Aunt Sally fashion, that this greater incidence of angina induced by the effort of men engaged in more active work acts as an early warning signal, which leads to greater care and, therefore, to lower mortality; but the point breaks down on more careful consideration. Whether the coronary circulation is benefited by physical activity *per se* or by any of the individual factors associated with it, or is damaged by lesser activity or any associated factors, remains to be determined. Morris and his colleagues state that there seem to be occupational factors in coronary heart disease other than the physical effort of the job. Quite substantial differences in their experiences of coronary heart disease have been encountered between members of occupational groups when no question of a substantial difference in physical activity arises (general practitioners, for example, have been found to have twice the incidence of other doctors), but the nature of the other occupational factors is unknown. Such evidence as is available does not suggest that the problem is simply one of responsibility or of greater mental work, but many individual factors require much more investigation. Morris and his colleagues state that there is every reason to suppose that various physical, psychological and social factors operate together, and it is quite possible that unfavourable factors cluster in certain occupational groups, particularly those in the upper social strata.

THE COMMON COLD.

THE common cold is ubiquitous and causes an immense amount of misery and economic loss, but, from a laboratory point of view, it is a most unsatisfactory and elusive virus disease. Progress in its study has been hampered by the fact that man and the chimpanzee are the only known susceptible animals. Attempts to implicate bacteria in the primary causation have not been successful, although, as secondary invaders, they make the later stages of the cold more unpleasant, and there is abundant evidence of the viral nature of its causation. C. H. Andrewes, who has done a considerable amount of work on the subject, has reviewed the present position.[1] He states that since 1946 a team of workers have carried out intensive research on the common cold at the Harvard Hospital, Salisbury, England. Thousands of volunteers have been exposed to infection in many ways under controlled conditions. The most active nasal washings, filtered or not, produced colds in 55% of women and 43% of men. The incubation period was usually two to three days from the time of intranasal instillation of virus. Evidence was produced that a subject might be infectious before symptoms appeared. Chilling of the subject by various means could not be shown to produce colds or to lower resistance to a small dose of virus. There was evidence that colds in children spread more readily to others than did colds in adults. Adults in houses where there was a school-age child caught twice as many colds as others who lacked this regular house contact with children. Infected nasal secretions might very readily be widely spread around manually, but the cold virus did not readily survive drying. It was found that although persons used handkerchiefs which had been contaminated with nasal secretions from infected persons and then briefly dried, few of them developed colds. One great difficulty has been that until very recently it has not been found possible to grow the virus, but Andrewes and a group of colleagues[2] have shown that it can be serially propagated in cultures of human embryonic lungs, so the possibility of producing an attenuated virus to be used as a locally applied vaccine is closer. Natural infection leads to little or no lasting immunity, although the fact that only about one in five persons exposed to infection contracts the disease makes it likely that many people develop an immunity from repeated contact with small doses of virus. No form of treatment has been found useful for aborting or curing colds. Much has been made of antihistamine drugs for treatment and prevention, but recent work has proved that they are useless. Modern advice on local medication of colds is to leave the nose well alone; it is much easier to do harm than good.

A NEW ABSTRACT PUBLICATION ON CANCER.

ARTICLES on cancer now appear in great number and variety in a wide range of medical and general scientific journals, and it is practically impossible for a worker in any part of this field to become aware of all the articles on the subject that may concern him. It is of interest, therefore, to learn that a new section (Section XVI) has now been added to the series published by Excerpta Medica, under the title "Cancer (Experimental and Clinical)". The publication of this section has been made possible by grants authorized by the National Cancer Institute of the Public Health Service and the American Cancer Society Incorporated. The new cancer section, like the existing fifteen sections prepared by Excerpta Medica, is to be published monthly. It will contain 700 to 800 pages of abstracts each year, including a monthly index of authors and a classified subject and authors' index with each yearly volume. The subscription rate for Volume I, which is to consist of six monthly issues, from July to December, 1953, is five dollars, or the equivalent in local currency. Volume II, 1954, and succeeding volumes will consist of twelve monthly issues and will be priced at ten dollars a year. The abstracts are grouped according to the following subjects: history, general theories regarding the origin of cancer, statistics, cancer control, experimental cancer research, general morphological pathology, metastasis, tumours in animals and plants, diagnostics, general aspects regarding treatment, and then the various anatomical regions of the body. The information made available in this way should be a great convenience to workers in cancer research and help to obviate unnecessary duplication of work in the various research centres throughout the world.

[1] *Brit. M. Bull.*, Volume IX, Number 3.
[2] *Lancet*, September 12, 1953.

Abstracts from Medical Literature.

OPHTHALMOLOGY.

Suction Anterior to the Iris and Narrow-Angle Glaucoma.

ADOLPH POSNER (*Am. J. Ophth.*, February, 1953), while admitting the factor of pupillary block in the production of acute glaucoma, advances a new theory of the mechanism of narrow-angle glaucoma. He thinks that the initiating factor responsible for the rise of tension in narrow-angle glaucoma is the relatively more rapid outflow of aqueous through the corneoscleral trabeculum as compared with the inflow into the chamber angle. In an eye with a shallow anterior chamber or narrow angle, the iris may come so close to the cornea that, in effect, it divides the anterior chamber into two zones: a central zone, which maintains normal relationships with the posterior chamber, and a peripheral zone. The two zones are normally in communication with each other, but certain conditions, such as dilatation of the pupil, congestion or œdema of the ciliary body and swelling or displacement of the lens, may cause contact between the iris and the cornea. The flow of aqueous into the peripheral zone is so retarded that it cannot keep pace with the outflow through the canal of Schlemm, and as a result the iris bulges forward to fill the space previously occupied by the aqueous. The iris presses against the trabeculum and obstructs the outflow; the intraocular pressure now rises. Miotics act by breaking the suction which holds the iris in contact with the trabeculum. Once the suction is broken, the entire anterior chamber deepens and the angle opens. With regard to surgical measures, peripheral iridectomy is superior to complete iridectomy; and when a fistulizing operation is desired, peripheral iridencleisis is effective. When the tension is normal between attacks, peripheral iridectomy is the operation of choice.

Experimental Herpes Simplex.

ALSON E. BRALEY (*Am. J. Ophth.*, December, 1952) discusses *herpes simplex* in relation to the eye. He recognizes four clinical types of infection, namely, acute primary *herpes simplex* blepharo-kerato-conjunctivitis, self-limited recurrent dendritic ulcers of the cornea without conjunctivitis, dendritic ulcer of the cornea which progresses to the development of disciform keratitis and uveitis, and chronic keratitis with some dendritic ulcer character with active and healed areas, which he calls *keratitis metaherpetica*. The acute primary kerato-conjunctivitis is sudden in onset with œdema of the lids and bulbar conjunctiva. Blisters develop on the lid border, and a preauricular lymph node is involved. The cornea shows a dendritic figure. The disease runs a course of fourteen to eighteen days, and healing occurs with only slight corneal scarring. It occurs usually in the teen ages. The second type, that of recurrent dendritic ulceration, is the commonest form of infection. The author believes that in these people the virus is present in the conjunctiva all the time and that it is released to produce a dendritic ulcer under appropriate conditions. One source of stimulus is the action of radiant energy on the eye. Another potent stimulus producing *herpes corneæ* is a fever. The mechanism of release of the herpes virus is not well understood, but it has been shown that cortisone can upset the local immunity. Disciform keratitis may develop in any of the types of herpes. It is a type of collagen reaction, and is effectively treated by cortisone. When a herpes lesion becomes chronic, there is a roughening of corneal epithelium with or without an associated disciform keratitis. The zone around the previous dendritic ulcer shows great variation in the thickness of the epithelium. There may be multiple small bullæ in the epithelium, so that it resembles bulbous keratitis. In many areas the epithelium is absent, the eye is irritable and there is low-grade irido-cyclitis. In this lesion there is a good deal of dormant virus present in the cells. In the treatment of this chronic recurrent condition the interference phenomenon may be of some value—that is, the use of another virus to interfere with the virus in the cell, for example, repeated vaccination. Experimentally it is found that in tissue culture folic acid will protect cells from infection with herpes virus, but it has no value in the treatment of clinical disease.

Present Status of ACTH, Cortisone and Hydrocortisone in Ophthalmology.

D. M. GORDON *et alii* (*Brit. J. Ophth.*, February, 1953) summarize present knowledge on the clinical use of ACTH and cortisone in ophthalmology. They state that cortisone used topically is highly efficient in treating superficial ocular disease. Although there is some intraocular absorption of topically applied cortisone, this amount may be insufficient to control an acute intraocular disease, but may be sufficient for its secondary control. Subconjunctival injections of cortisone are often sufficient in cases of anterior uveitis, but are less frequently so with posterior segment disease, and an initial course of systemic therapy may be essential. For inflammatory disease of the posterior globe or severe anterior segment disease which has not responded to topical or subconjunctival cortisone, systemic therapy is indicated. If the patient is ambulatory, oral or intramuscular administration of cortisone or intramuscular administration of ACTH in a long-acting vehicle is satisfactory. If the patient requires admission to hospital, the use of ACTH by prolonged intravenous drip administration is the most efficient weapon available; 25 milligrammes of ACTH given intravenously in eight hours is equivalent to 300 milligrammes of cortisone. In the treatment of chronic disease the principle is that once severe manifestations of the inflammation have been suppressed by intensive systemic treatment, they can be held in a state of suppression, frequently by less intensive therapy. The authors give their patients with chronic inflammatory disease a course of intravenous ACTH therapy and then work out a maintenance dose of the hormone. As a rule local therapy does not suffice, at least in the early stages. As sterilization of cortisone drops is difficult, the authors use sulphacetamide with the cortisone to combat infection and render the solution sterile. For local allergies the authors recommend cortisone and an antihistaminic, and thereby weaker solutions of cortisone are made efficient. The chief contraindication to the use of these agents is infection due to various viruses. Hydrocortisone (compound F) has been employed in 60 cases in the same manner and dosages as cortisone. Given systemically it appears to be more effective than cortisone in lesser concentrations, and given subconjunctivally it provokes less reaction than cortisone. The authors suggest that it will probably replace cortisone.

Retinal Detachment with Retinal Tears in Retrolental Fibroplasia.

H. A. LERNER (*Arch. Ophth.*, July, 1953) describes two cases of retrolental fibroplasia in which retinal detachment was associated with a tear. In both cases surgical treatment was undertaken to seal the tear and to drain subretinal fluid. Subsequent examination showed the tears to be sealed and the retina flat in position. The author stresses the importance of repeated and long-continued ophthalmoscopic examination of all premature infants with the hope of discovering the pathological process at a time when it can be checked by surgical means.

Cat-Scratch Disease and Parinaud's Oculoglandular Syndrome.

J. V. CASSADY AND CARL S. CULBERTSON (*Arch. Ophth.*, July, 1953) discuss the ætiology of Parinaud's oculoglandular syndrome and produce evidence and report four cases to support the contention that the ætiological agent is probably that of cat-scratch disease. In the four cases the results of all investigations were negative except that of the intradermal test with cat-scratch antigen which was positive. The authors state that transmission of Parinaud's syndrome is not from patient to patient. Cases are usually sporadic and usually only one member of a family is infected. The clinical features occurring in Parinaud's oculoglandular syndrome are quite similar to those occurring in the cutaneous form of cat-scratch fever. Parinaud's oculoglandular syndrome is a manifestation of cat-scratch fever which differs in that the conjunctiva is the site of primary infection, the initial lesion being one of conjunctivitis with granulation followed by regional lymphadenitis. Reaction to the skin test remains positive for a considerable period; how soon it will become positive in the oculoglandular syndrome is unknown.

OTO-RHINO-LARYNGOLOGY.

Surgical Treatment of Bilateral Abductor Paralysis.

A. A. SCHEER (*Arch. Otolaryng.*, February, 1953) states that there have been many approaches to the surgical treatment of bilateral abductor paralysis. All have the same objectives—to reestablish an adequate airway and to obtain the maximum speaking voice. The attainment of these objectives has not been achieved with any real ease or calculation. Easy visualization of the operative field is

desirable. The most direct approach to the vocal cords and arytænoids is by means of the laryngofissure technique. The operation may be performed with the use of local anæsthesia. A preliminary tracheotomy is necessary. After the larynx is opened and held apart, a fine "3-0" stainless steel suture is put through the selected vocal cord in front of the vocal process. The suture is passed from below upwards, just beneath the mucosal surface. Its free ends are then passed out through the thyroid cartilage along a hollow needle which is thrust from without inwards through a separate incision overlying the posterior border of the thyroid ala. This suture is used later to hold the vocal cord in a position of optimal abduction. The arytænoid cartilage is resected through a small vertical incision in the mucous membrane overlying its anterior surface and extending from the crico-arytænoid joint to the upper pole of the arytænoid. A single small suture is used to close the arytænoid bed, and the mucosal incision is then closed very accurately. The free ends of the steel suture are tied down on the outside of the thyroid cartilage, the vocal cord being thus fixed in abduction.

The Bronchus and Pulmonary Tuberculosis.

F. H. YOUNG (*Edinburgh M. J.*, February, 1953) states that it is only in the past fifteen to twenty years that tuberculous bronchitis has begun to be recognized as an important part of pulmonary tuberculosis. Bronchial disease can explain many previously inexplicable phenomena. Bronchoscopy, however, is fraught with pitfalls. What can be seen are only changes in the mucous membrane and dilatation of the bronchi and alteration in their lumina. The cut stumps of the bronchi of resected portions of lung tissue have been examined. In these cases, although it might be assumed that the area through which the surgeon made his section was bronchoscopically normal, yet in 93% of 96 cases definite evidence of tuberculosis was found in the walls or in the mucous membrane adjacent to the sectioned bronchus. Of 80 cases of resection in which bronchoscopic examination findings had been reported as normal, gross bronchial stenosis was found at subsequent examination in a very high proportion. It has been found that the incidence of tuberculous bronchitis increases with the severity of parenchymal disease. Infection may be by direct implantation in the bronchial mucous membrane, by lymphatic extension or from ulceration of tuberculous glands. Healing may be without effect, or bronchiectasis or stenosis of the bronchus may be brought about. Cases may occur in which there is gross tuberculous bronchitis without cavitation and without evidence of tuberculous disease in the lung parenchyma. One of the methods by which cavities are naturally assisted to heal is through bronchial stenosis. Bronchial abnormality, either stenosis or thickening of the bronchial carina, associated with œdema was found in a high proportion of cases with slowly absorbing pleural effusion. Bronchial disease is responsible for a high proportion of pneumothorax disasters. Ballooned cavities are thought to be influenced by bronchial disease, but the exact mechanism is not settled. True tuberculous bronchiectasis may occur, although the proportion of cases may not be high. Some tuberculous cavities are not really holes in the lung produced by tissue destruction, but are really bronchial abscesses associated often with proximal bronchial stenosis. Bronchoscopic examination need not be carried out in every case and may occasionally cause spread of the disease. The indications for bronchoscopy in cases of pulmonary tuberculosis are as follows: (*a*) hæmoptysis, especially if repeated without X-ray evidence of cause, (*b*) repeated positive sputum findings without demonstrable cavities, (*c*) spasmodic cough, (*d*) wheeze—localized, especially at night, and shifting on movement, (*e*) persistent pleural effusion, (*f*) appearance of considerable fluid in an artificial pneumothorax, (*g*) lobar or segmental atelectasis or an abnormally small lobe, (*h*) sudden consolidation of a lobe under collapse, (*i*) spread of disease from a lesion apparently controlled, (*j*) blocked or ballooned cavity.

Intraepithelial Carcinoma of the Larynx.

F. ALTMANN, I. GINSBERG AND A. P. STOUT (*Arch. Otolaryng.*, August, 1952) state that laryngeal intraepithelial neoplastic growths without invasion, which are comparable to those found in many other mucous membranes and in the skin, have passed almost unnoticed by laryngologists and pathologists. At the Presbyterian Hospital, New York, in the decade 1940-1950, 57 of 312 histologically diagnosed laryngeal epitheliomata showed intraepithelial changes such as warranted the diagnosis of intraepithelial epithelioma. Nineteen of these showed some degree of infiltrative growth; the remaining 29 cases (or 9·3% of the total number of laryngeal cancers) showed only intraepithelial cancerous changes, in one or several subsequent biopsies. The mean ages of the patients in the infiltrative and in the intraepithelial cases were approximately the same. Histologically the growth originates from squamous epithelium and shows essentially the same cellular characteristics as ordinary squamous cell epithelioma. The main distinguishing feature is its peculiar spread within the epithelium towards the periphery instead of infiltrating through the basement membrane into the underlying connective tissue. The condition is regarded as true cancer from the beginning and not as a precancerous condition. The great majority of the tumours originate from the vocal cords. They appear as circumscribed or more diffuse greyish, whitish or more reddish thickenings of the vocal cords, with a smooth or slightly granular surface. The anterior part or the middle third is most frequently involved. Extension may occur across the anterior commissure into the other cord and down into the subglottic space. In some instances a more diffuse thickening of the entire cord is observed, but the mobility of the cord is unimpaired in all instances. The average duration of symptoms in all cases, both infiltrative and otherwise, is about six months. A review of the clinical course shows that this is a less malignant process than infiltrating cancer. The question arises whether the same radical procedures as for frankly invasive tumours are really necessary. Radiotherapy should be seriously considered in cases without any or with only limited invasion. The authors report that in two cases in which radiotherapy was used a diffuse infiltration of the whole cord was seen without impairment of mobility. Radiotherapy was used in two other cases with diffuse changes in both cords. One patient has been symptom-free for twelve months, and one died of cardiac failure after thirty-six months. If treated surgically these patients would have required laryngectomy.

Sympathetic Surgery for Ménière's Disease, Tinnitus and Nerve Deafness.

E. R. GARNETT PASSE (*Arch Otolaryng.*, March, 1953) states that the thesis has arisen from consideration of the hypothesis that neuro-vascular disturbances, the result of an autonomic imbalance, could be the ætiological factor in some cases of Ménière's disease, of tinnitus, and of nerve deafness. It has been demonstrated that there may be hyperexcitability of the labyrinth after administration of drugs which stimulate the sympathetic and hypoexcitability after administration of depressor drugs, and that vasodilatation produces hypoexcitability and vasoconstriction hyperexcitability. Stimulation of the cervical sympathetic produces reduction in calibre of the intracranial vessels. The author has observed gross dilatation of the vessels of the membranous canal as the result of a procaine block of the stellate ganglion during a fenestration operation. There may be several different mechanisms which produce the symptoms of Ménière's disease. Retention of fluid and endolymphatic distension may be influenced by variations in capillary blood flow and permeability. Stellate ganglion block with 1% procaine solution is used as a diagnostic test in the selection of cases. Within a minute the blood vessels of the tympanic membrane and of the conjunctiva become dilated. The injection is a relatively simple procedure and does not necessitate admission to hospital. Adrenaline should not be used with the procaine solution. The presence of cervical osteoarthritis should be excluded by X-ray examination, as it has been found that such a condition of the sixth cervical intervertebral joint may be associated with tinnitus and vertigo, the symptoms being relieved by a procaine injection around the neural grooves of the fifth and sixth cervical vertebræ. The relief is made permanent by preganglionic upper dorsal sympathectomy. If the tinnitus is subdued, there is also an improvement in the hearing, especially when comparative testing is made with the articulation score in speech audiograms. Resection of the stellate ganglion, together with stripping of the vertebral artery and preganglionic isolation of the second and third dorsal ganglia, is the procedure carried out by the author. Ménière's attacks have been stopped permanently in 62% of cases, with relief in 35%. The results have been remarkable in a number of cases of idiopathic nerve deafness, 60% to 70% of patients showing a gain in hearing of over 10 decibels. Tinnitus was relieved in 50% of cases.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on June 18, 1953, at the Royal Alexandra Hospital for Children, Sydney. The meeting took the form of a series of clinical demonstrations by the members of the honorary medical staff of the hospital. Part of this report appeared in the issue of December 12, 1953.

Resistant Rickets.

DR. S. E. J. ROBERTSON showed a female patient, aged five and a half years, whose birth, neonatal period and development during infancy had been normal. She had been breast fed until the age of nine months, had received supplements of vitamin *D* and was often exposed to the sun. When aged seventeen months she was thought to have unduly bowed legs, and when aged twenty-two months she was diagnosed as suffering from rickets. She was given 2000 units of vitamin *D* daily and kept in night splints for three years without improvement. There was no history suggestive of renal or liver disease, of malabsorption of fat or of hypocalcæmia, nor was there any family history of rickets or any disturbance of growth.

On physical examination of the patient, it was seen that her growth was slightly retarded, but her body proportions were normal for her age. Her muscle tone was less than normal and her abdomen was slightly protuberant. Her legs were severely bowed, the bowing affecting both femora and tibiæ; but there was no enlargement of the epiphyses. Pronounced radiological changes of rickets were present in the epiphyses of the radii, ulnæ, tibiæ and fibulæ.

The patient was investigated at the hospital, and at the Clinical Research Unit of the Royal Prince Alfred Hospital by the courtesy of Dr. C. R. B. Blackburn. Her blood was found to have a low inorganic phosphorus content, a high alkaline phosphatase content, and normal calcium, urea nitrogen, bicarbonate, chloride, sodium and potassium contents. Her urine contained no reducing substances, no abnormal amino-acids, no excess calcium and no cells or casts. The results of urine concentration tests and the creatinine clearance were normal. Her response to ingestion of ammonium chloride showed normal tubular function. No improvement in the radiological picture followed very large doses of vitamin *D* administered parenterally, or the exhibition of dehydrotachysterol. It was thought that the patient's rickets was due to failure of utilization of vitamin *D* at the growing bone ends.

Dwarfism.

Dr. Robertson's second patient was a female, aged eight years, who had been first examined in October, 1952, with complaints of being small since the age of three years and of rapidly increasing weight over the previous twelve months. After a normal birth she had developed normally in infancy, and she had progressed normally at school. There were no symptoms to suggest hypothyroidism or an intracranial lesion in the vicinity of the pituitary or hypothalamus. Her previous health had been good, and she had had no infectious fevers before the age of three years. None of her relatives had growth disturbances.

On physical examination of the patient, her measurements were as shown in Table I. Dr. Robertson pointed out

TABLE I.

Observation.	Average for Height.	Average for Age.
Weight, 51·25 pounds	34·0 pounds.	49·5 pounds.
Height, 39·6 inches	—	47·9 inches.
Span, 39·0 inches	38·1 inches.	47·1 inches.
Upper measurement, 20·6 inches ..	22·0 inches.	24·7 inches.
Lower measurement, 19·0 inches ..	17·6 inches.	23·2 inches.
Ratio, upper : lower, 1·25	1·25.	1·06.
Head circumference, 19·5 inches ..	19·7 inches.	20·3 inches.
Chest circumference, 24·75 inches ..	20·7 inches.	23·0 inches.
Abdomen circumference, 24·75 inches	19·5 inches.	20·7 inches.

that the patient's height, span and head circumference were low, that the ratio of upper to lower measurements was normal for her height but reduced for her age, indicating infantile proportions, and that her obesity had caused increase in chest and abdominal circumferences. She had a small nose with immature features. The increased weight was due to excessive adipose tissue, which was general in distribution and firm in consistency, and it did not suggest an endocrine deficiency. The peripheral circulation did not seem deficient. She had only 16 deciduous teeth. Radiological examination revealed an osseous age of three years, with no epiphyseal dysgenesis or abnormalities of bone structure. No evidence was discovered of any occult chronic disease which might have been retarding growth. The serum cholesterol content was 180 milligrammes *per centum*, and the basal metabolic rate −13%. Neither the ratio of the urinary uric acid to creatinine and the level of circulating eosinophile cells after the injection of adrenaline, nor the blood sugar curve after the intravenous injection of insulin, suggested pituitary deficiency. It was decided that there was a strong possibility of thyroid deficiency.

Dr. Robertson said that since February, 1953, the patient had been treated in the out-patient department with increasing doses of thyroid extract. Owing to infrequent attendances the following dosage had been employed: from February 2 to 18, one-eighth of a grain daily; from February 18 to March 25, half a grain daily; from March 25 to April 29, one and a half grains daily; and from June 10, two grains daily. When the patient was examined on June 10 she had no symptoms or signs of thyroid over-dosage. She was not doing well at school, although she was trying hard. Her two permanent lower central incisors had just erupted. Her serum cholesterol content was 160 milligrammes *per centum* and her osseous age was three years. Her measurements were as follows: weight 46·5 pounds, height 42 inches, upper measurement 22·75 inches, lower measurement 19·25 inches, head circumference 19·9 inches, chest circumference 23·75 inches, abdominal circumference 23·5 inches.

Dr. Robertson said that from the changes which had occurred with thyroid therapy in the last four months—namely, the fall in serum cholesterol content, the eruption of two permanent teeth, the reduction of obesity and the increase in height—it seemed that the child was responding to thyroid extract. However, radiological studies revealed no significant change in the number of epiphyseal centres. It was planned to increase the dosage of thyroid to tolerance and to make further observations in the next few months.

Diabetes Mellitus with Insulin Resistance.

Dr. Robertson's next patient was a male, aged seven and a half years, who had been diagnosed as having *diabetes mellitus* in April, 1952. During 1952 his condition was kept stable with a moderate dose of soluble ordinary insulin twice daily except when he developed mild infections, when his dose had to be increased and the injection given three times daily. In December, 1952, symptoms of diabetes recurred, and he began to pass very large amounts of sugar in his urine without apparent cause. In January, 1953, he was admitted to the Royal Alexandra Hospital for Children for stabilization. No abnormality was found to account for any resistance to insulin, and it was necessary to increase his insulin dosage gradually until his condition was eventually brought under control in April, 1953, when he was having protamine zinc insulin, 100 units in the morning and 12 in the evening, and ordinary insulin, 104 units in the morning and 100 in the evening, each given by separate injections. After that his requirement for insulin began to fall rapidly. On May 27 he was having protamine zinc insulin, 32 units in the morning, and ordinary insulin, 64 units in the morning and 36 in the evening, and on that dosage his blood sugar levels over twenty-four hours were verging on hypoglycæmic levels. Since that time his insulin dosage had to be increased owing to minor infections, and recently he had gone into a coma, his carbon dioxide combining power being 12 volumes *per centum*. Dehydration was not severe in that episode and was easily relieved; but he required enormous doses of insulin to reduce his blood sugar to normal levels.

Dr. Robertson said that the insufficiency of insulin, which was fundamental in the pathogenesis of *diabetes mellitus*, might be due to the following factors: (i) an absolute decrease in available insulin (severe intrinsic pancreatic disease and total pancreatectomy); (ii) an increased need for insulin due to its increased utilization (over-feeding, obesity and hyperthyroidism); (iii) an increase in the rate of insulin destruction; (iv) a decrease in the responsiveness of the enzyme systems affected by insulin (endocrine factors such as purified growth hormone, crude anterior pituitary extract, adrenal cortical steroids and ACTH and liver disease); (v) the production of insulin antagonists or neutralizing agents. Except for the first category—an absolute decrease in the amount of available insulin such as

obtained in total pancreatectomy—it was obvious that all the other mechanisms responsible for the clinical syndrome of diabetes might give rise to a state of insulin resistance or refractoriness. Differences in insulin tolerance of several hundredfold were observed among certain strains of mice, while non-diabetic human beings might tolerate as much as 1000 units without hypoglycæmia. Such normal variations should also be expected among diabetic patients. Furthermore, no standard ratio of insulin need to food intake could ever be established except on an individual patient basis, and even then severe fluctuations obtained, as had been described previously.

Dr. Robertson went on to say that just as the causes of diabetes remained unknown, so did the origins of "insulin resistance" in the majority of instances. Certain obvious factors occasionally induced unresponsiveness to insulin, including allergy to insulin, acute infections, diabetic acidosis, liver disease, and hyperfunction of the thyroid gland, the anterior lobe of the pituitary and the adrenal cortex. All those disorders, however, might also occur in diabetes without producing that phenomenon. Consequently, most reports on the subject revealed only fruitless results from elaborate investigations for hormonal abnormalities, neutralizing antibodies to insulin, hepatic dysfunction and so forth. Similar "insulin sensitivity" usually defied elucidation except in the rare case of total pancreatectomy, and in the even rarer association of diabetes with Addison's disease, myxœdema and anterior pituitary hypofunction. The average "brittle" case presented marked lability between hypoglycæmia and ketosis, often with a dosage limited to but 20 to 40 units a day. Such patients taxed the ingenuity of the physician in obtaining a fixed regimen, and might require a flexible programme of several daily injections of regular insulin. The treatment of "insulin resistance" consisted solely in the administration without timidity of as much insulin as was needed to insure adequate utilization of the diet. Non-specific therapy directed at the remote possibility of pituitary and thyroid over-activity had been attempted by means of intense X irradiation of either gland or by the induction of myxœdema by ablation or the administration of radioactive iodine, with equivocal results from both. "Insulin resistance" was usually a transitory phenomenon, most patients returning to the previous insulin requirement after several months. In rare instances complete remission of diabetes followed a period of "insulin resistance".

Dr. Robertson said that the outlook for the patient under discussion was uncertain; the cause of his "insulin resistance" was quite unknown. It was possible that his requirement might continue to increase, but at the moment he required close observation with laboratory control to maintain him in equilibrium.

Fits in Infancy.

DR. L. R. RAIL showed several patients suffering from seizures, to illustrate the correlations between the clinical state and the electroencephalographic findings.

Epilepsy of Idiopathic Origin.

The first patient was aged ten months. Dr. Rail said that in this case the birth had been normal; there were no neonatal disturbances. Development and mentality had been within normal limits until the tenth month. At that stage four generalized convulsions occurred associated with febrile episodes. There was a family history of epilepsy. The electroencephalographic record was normal. That was regarded as a good prognostic sign. The diagnosis was most likely epilepsy of idiopathic origin rather than that associated with brain damage or developmental defect.

Myoclonus Associated with Birth Injury or Developmental Defect.

Dr. Rail's second patient, aged ten months, had been delivered by Cæsarean section; no obvious disability had been observed until the third week. At that stage attacks of jerking movements of the arms began, which were associated with very brief loss of consciousness. No major convulsions had occurred.

On examination, the child was of normal physical appearance. Attention could not be gained by visual or auditory stimulation. There were no organized voluntary movements of the arms, the tone was generally reduced and the reflexes were absent. The clinical state suggested brain damage or developmental defect. The electroencephalogram was grossly disturbed.

Birth Injury Associated with Fits and Mental Defect.

Dr. Rail next showed a male child, aged eighteen months, whose heredity and family history were normal. The child had had a prolonged and difficult birth, although the mother's pregnancy was normal, and was treated in an incubator for over a week. The institution of feeding was difficult. The child's attention had always been defective, and he took little notice of his surroundings. Seizures began when he was aged one month. Mass myoclonic jerking and "jacknife" seizures were frequent in the first few months. They were replaced by generalized convulsions by the sixth month. Various forms of generalized seizures had occurred since, sometimes four or five in a single day.

On examination of the child, his attention could not be secured with visual stimuli, but he responded to painful stimulation and loud noises. His limbs were flaccid and the tendon jerks were absent; no organized voluntary movement was apparent. The electroencephalogram showed gross slow wave and spike dysrhythmia.

Minor Seizures.

The next patient shown by Dr. Rail was a girl, aged twenty months. An aunt was epileptic. The child had been born normally after a normal pregnancy, and had developed normally. At the age of eleven months she began to have attacks in which she fell to the ground limp and unconscious; she had had no convulsions.

On examination, the patient appeared a normal child for her age, with no neurological defects. The electroencephalographic record was within normal limits.

Brain Damage with Measles Encephalitis.

Dr. Rail's next patient was aged nine months, and had been normal until an attack of morbilli at the age of three months; after this the child lost interest in toys and in the environment generally. Seizures, consisting of the rolling of the eyes upwards with drawing up of the arms and legs, had begun after the measles, and had occurred once or twice per day since. An electroencephalogram taken during natural sleep was grossly abnormal and was consistent with brain damage associated with measles encephalitis.

Breath-Holding Turns.

Another patient shown by Dr. Rail was aged nine months, and had a strong hereditary taint of epilepsy. At the age of seven months she began to have typical breath-holding turns. No abnormalities were detected by physical examination or in the electroencephalogram, which was partly a sleep recording.

Peripheral Neuritis in a Child.

Dr. Rail finally showed a male baby, aged five months, whose heredity was normal and whose family history was clear. The child had been born normally after a normal pregnancy. The mother had contracted influenza when the child was five weeks old, and the child contracted "tonsillitis"; it was treated with penicillin and cleared up in a few days. When the baby was aged about three months, the mother noted that the baby did not move his arms normally. They tended to hang down by his side with the fingers flexed. As the weeks went by some return of function occurred, and at the time of the meeting the child was able to lift his arms to a right angle, but could not grip objects well with either hand. There was no disturbance of proprioception or of the sensory functions. On being questioned, the mother said that the baby moved his legs, but did not move his ankles or toes. He was breast fed.

On examination, the baby was seen to be a well-nourished child of intelligent aspect. He had no skin lesions. His attention could be readily attracted, and he would reach for objects, but was unable to grip well with his fingers. He moved his legs, but there was no movement of the feet or toes. The tendon reflexes were absent. The strength of the proximal upper limb muscles was reduced, but the main disability was in the extension and flexion of the fingers.

A blood count gave normal results. The arsenic and lead contents of the mother's urine and hair were normal. A Schick test indicated that the child had been in contact with diphtheria (*"Schick-negative"*).

The condition was regarded as diphtheritic polyneuritis.

Rheumatic Disease and Cœliac Disease.

A demonstration was arranged by the Institute of Child Health illustrating certain features of rheumatic disease in childhood and of cœliac disease in infancy.

The features of rheumatic disease illustrated were the following: (i) American and British ideas on the remote prognosis of acute juvenile rheumatism. (ii) Current ideas about salicylate and hormone therapy. A graph illustrating

the treatment of a child with salicylates and cortisone was shown. (iii) Recent ideas on prophylaxis.

With regard to cœliac disease, the following features were illustrated: (i) Diagnostic difficulties, with particular reference to radiological changes in the alimentary tract, non-flocculating barium being used. (ii) The place of the gluten-free diet in diagnosis and treatment.

(To be continued.)

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

CONTAGIOUS DISEASE ON THE CONVICT SHIP "PORTLAND".[1]

Medical Department,
Sydney,
June 26, 1833.

The Honorable,
The Colonial Secretary.

Sir,

I have the honor to acquaint you for the information of the Governor that I proceeded aboard the convict ship "Portland" agreeably to His Excellency's instructions and was informed by the surgeon of the ship that the guard embarked on the 28th of December since which it appears by the sick list ninety-three cases of disease have been treated on board, eight of which were cholera morbus and of that number three died. The last case of cholera occurred on the 6th of March which day the ship anchored at Lisbon but not being permitted to communicate with the shore proceeded to sea the following day.

Several cases of Typhus Fever occurred during the voyage and two of the convicts and one of the crew died of that disease. Three other deaths took place from Pulmonary Consumption, Dysentery and Dropsy. The last of these was on the 22nd of May and since that date all on board have been quite healthy there being at present only two slight cases under treatment. The Surgeon reported that every precaution had been taken to clear and ventilate the ship and there does not appear to be any contagious disease among the convicts guard or crew consequently there will be no necessity for placing the ship in Quarantine.

I have, etc.,
JAS. BOWMAN.

Correspondence.

THE MODERN TREATMENT OF BENIGN PROSTATIC OBSTRUCTION.

SIR: In his paper on "The Modern Treatment of Benign Prostatic Obstruction", Mr. Ian Hamilton (*M. J. AUSTRALIA*, November 21, 1953) makes it clear that he does not like the operation of endoscopic resection.

In support of his opinions, however, he makes some statements which might be misleading to those who are unacquainted with modern urological methods. For example, to say that "the complications of this endoscopic operation, of which incontinence is the greatest, are such that better methods have been sought" would seem to imply that it is a recent fad which is falling into disrepute. This is not so. Incontinence of urine is a rare complication of endoscopic resection.

In the 1930's, as a result of enthusiastic salesmanship, an enormous number of resectoscopes was sold in America to doctors, many of whom were untrained in urology, and who imagined that endoscopic resection was an easy method of dealing with bladder-neck obstruction. Disillusionment soon came, and the country rocked under a wave of hæmorrhage, incontinence and ruptured bladders. Consequently the sale of incontinence clamps did, as Mr. Hamilton states, rise steeply. But all that happened twenty years ago.

The operation finally came to rest in the hands of those who were qualified to perform it. There is no question as to whether endoscopic operation is, or is not, superior to the open approach. The trained urologist is skilled in both, and chooses the operation to suit the case, and not because of any personal preference.

Yours, etc.,
D. COLEMAN TRAINOR.

135 Macquarie Street,
Sydney,
December 7, 1953.

PINK DISEASE.

SIR: Any approach to the manifestation we call "pink disease" is justified if it adds something to the therapeutic cupboard. So far we have been relying on vitamin *B* and liver, which are apparently replacements while "nature effects a cure". In the various animadversions on "pink disease", we in the periphery have been "blinded by science", all the research energy being expended on the terminal conditions of exhaustion and malnutrition.

The following are among the important facts established by collected reports on the subject:

1. "Pink disease" occurs in districts. This is a "district" with a distinct "intermarried" flavour.

2. It occurs in families. There have been four families here that I know of with two cases in one family.

3. Babies do not die of "pink disease". They die of intercurrent infections, exhaustion and malnutrition; that is to say, there is no known pathology.

4. Recovery is always complete. In other words, again there is no known pathology.

5. The signs and symptoms are essentially those of an allergic condition, or at any rate they closely resemble these signs.

The following is an interesting and typical case. Baby H. was, according to the mother, always "a good baby". On June 9 at seven months he was at a birthday party with a cold. He became "irritable and unmanageable". After this he had "one cold after another". He developed a "wheeze in the chest". As the grandfather was an asthmatic, this alarmed the mother. In the middle of July she brought him to me about the cold and the "wheeze". He was given "Dulcet Duozine" and a cough mixture. This was of no value. I believe they made him worse.

Early in August he developed a herpes round the lower lip. In the middle of August the mother brought him to me, convinced that he had "pink disease". She had assisted in the nursing of his infant cousin, who had "pink disease" for about ten months. He had, by then, the usual array of "pink" signs and symptoms—misery, hypotonia, anorexia, insomnia, head-banging, cold feet, a burning feeling, rashes, "screwing up his eyes" *et cetera*. He was put on the recognized treatment—concentrated vitamin, liver and so on. He showed no improvement over a period of six weeks.

On October 5 he was given an injection of "Pernexin" (vitamin B_{12}) in the deltoid. This upset the applecart completely. The deltoid pain from the intramuscular injection gave all parties a night of misery. A trained nurse who had considerable experience nursing these cases at Renwick Infant Hospital, Summer Hill, recorded the fact that the child was in an advanced state of "pink disease" and warned the parents that they could look forward to six months of patient nursing and endurance.

I then suggested to the parents that the idea I had for some time that the condition was essentially a severe allergic manifestation was entitled to a test. He was given "Elixir" Benadryl" in half-drachm doses four-hourly. The result was immediate and dramatic. No other sedative was required that night. He slept peacefully without his usual chloral and bromide.

Within a few days he was apparently a normal happy infant. A fortnight after this treatment had been started the nurse who had seen him on October 6 was vividly impressed with the change from the severe manifestations (particularly the whining and hypotonia) to the appearance of a perfectly happy and normal infant of eleven months. During that fortnight he had two injections of vitamin B_{12}, but I doubt if they had any other effect but that of reminding him of his other ordeals.

[1] From the original in the Mitchell Library, Sydney.

The mother gives the baby an occasional dose of "Elixir Benadryl" if she thinks he is getting "cranky". The only sign of importance that has appeared since has been what the mother calls a "heat rash" on his neck. One type of rash that appeared during his period of torment was a "solar urticaria"—a measly rash that appeared on exposure to the sun. He has been "cranky" occasionally when parted from the other children of the house.

What is "pink disease"? Take a dose of hay fever with its accompanying misery, burning eyes and naso-pharynx; a dose of the common cold with its accompanying skin action; one of the exanthemata; serum sickness with its feeling of intense burning all over; drug, plant and food allergy; milk "eczema"; and solar urticaria. Sort out the worst features of these allergic conditions and plant them in a baby surrounded by adults who "don't understand", and I fancy we would have at least a good imitation of the condition we call "pink disease".

If my assumption is fallacious, no harm is done, as "Elixir Benadryl" is a valuable sedative, and effective sedation is the greater part of the battle against a condition which is as distressing and exhausting to the harassed mother as it is to the pathetic little patient.

Yours, etc.,

MAURICE MATHESON.

Tumbarumba,
New South Wales,
December 3, 1953.

A REPORT ON AN INVESTIGATION OF A COUNTRY MIDWIFERY PRACTICE WITH THE PRELIMINARY RESULTS OF 300 CASES FOLLOWING IMPROVED OBSTETRICAL CARE.

SIR: Dr. L. O. S. Poidevin's comments on my high external version rate raises the question of unorthodoxy in one's personal methods of practice. Despite the fact that I have known that this is not accepted teaching, it is obvious that a single investigator will never have sufficient cases to accumulate convincing results in any one particular abnormality; he can only lower his gross mortality rate by constantly changing his methods; and I will no doubt cut down the number of versions in future. It would be of considerable assistance if the major teaching hospitals could annually publish a detailed statistical summary in one of the pages of THE MEDICAL JOURNAL OF AUSTRALIA of their current management of individual abnormalities, for example, version rate, manual removal rate, toxæmia rate, Cæsarean section rate, forceps rate et cetera. This would enable the individual in private practice to accurately gauge current trends in greater detail.

Yours, etc.,

K. J. CULLEN.

Busselton,
Western Australia,
December 3, 1953.

Obituary.

HAROLD JOHN RITCHIE.

DR. E. S. MEYERS writes: In company with some thirty or more students I became a member of the clinical school of the Sydney Hospital which had recently been established, ours being the third group of students to elect to go to Sydney Hospital. Those were far-off halcyon days when there were no wars, depressions or atom bombs. In due course we commenced duty in medical wards, and our lecturer in clinical medicine was Dr. Sydney Jamieson. We immediately discovered that Dr. Jamieson thought most highly of Dr. Ritchie, and although he was quite a recent graduate, often sought his opinion on various patients. We did not take long to observe that Dr. Ritchie was very keen and observant of patients in the wards; and I remember on one occasion, when we were examining a patient who, to our untutored ears, coughed much the same as any other patient would do, Dr. Ritchie, who had just left the ward, put his head around the corner and said: "That man has an aneurysm." This turned out to be the case.

The Calendar of the University shows that he graduated in 1911, and that he was not only the youngest honorary physician to the out-patient department, but the sole repre-

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED NOVEMBER 28, 1953.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	10(2)	5(4)	..	..	3(3)	..	..	..	18
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	1	..	..	..	..	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	1(1)	5(5)	6(5)	..	..	..	..	..	12
Diphtheria	7(5)	2(1)	2	..	1(1)	..	..	..	12
Dysentery (Bacillary)	..	..	4(3)	..	1(1)	..	..	..	5
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	1	..	..	..	..	..	..	1
Infective Hepatitis	..	8(1)	..	..	7(3)	..	..	..	15
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	3(1)	..	1	..	..	1	..	..	5
Ophthalmia	..	..	..	..	1	..	..	..	1
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	14(11)	4(4)	..	2(2)	3(3)	..	..	..	23
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	9(5)	1	..	56(41)	..	..	1	67
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	16(14)	25(20)	15(13)	1(1)	2(2)	..	..	..	59
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	1(1)	..	..	..	..	..	..	1
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	45(32)	45(36)	6(4)	7(5)	12(10)	4(1)	3	1	123
Typhoid Fever	2(2)	..	..	..	1	..	..	..	3
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

sentative on the medical side of the home graduates, as most of the clinical teachers at that time had graduated overseas. His out-patient classes were always instructive, and there was much to be learnt. We were impressed by his observation of patients and the deductions he drew from the observations after the manner of Sherlock Holmes. I subsequently became house physician to him when he had the care of a few in-patients and found he was extremely well read. I believe, while waiting for patients in Macquarie Street, he had read "Osler" thirty-seven times, and he was undoubtedly a great exponent of the Oslerian tradition. As time went on he eventually achieved high office and was one of the leading members of the National Health and Medical Research Council, although he had never taken a higher degree or diploma. He had all the attributes of a great physician.

He visited Brisbane on many occasions and in 1935 was one of the lecturers at the refresher course held annually. The lectures that he delivered were of great value to the general practitioner—the titles being "Digitalis in Medication", "The Various Forms of Acute Nephritis with Special Reference to the Prognosis" and "The Dyspepsias". On this occasion he took the opportunity to visit many old friends of his student days.

Dr. Ritchie was a forerunner of very many excellent Sydney graduates who were to instruct later generations of medical students, and those who had the good fortune to serve under him were shown by precept and example the best that medicine has to offer.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments have been promulgated in the *Commonwealth of Australia Gazette*, Number 76, of November 26, 1953.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

The probationary appointment of the following Flight Lieutenants is confirmed: G. S. Radford (025626), D. B. G. Bailie (039232).

Deaths.

THE following deaths have been announced:

BARTLEY.—Joseph Francis Bartley, on December 2, 1953, at Rosebud, Victoria.

MACLEOD.—Kenneth John MacLeod, on December 6, 1953, at Caulfield, Victoria.

Medical Appointments.

Dr. J. S. B. Lindsay has been appointed Senior Medical Officer, Mental Hygiene Branch, Department of Health, Victoria.

Dr. F. R. Hone has been appointed a member of the Council of the Institute of Medical and Veterinary Science, Adelaide, on the nomination of the Council of the University of Adelaide.

Dr. C. O. F. Rieger has been appointed a member of the Medical Board of South Australia.

Dr. H. J. C. Edmonds has been appointed Chief Medical Officer, Mental Hygiene Authority, Department of Health, Victoria.

Dr. B. B. Joyce (nominated by the Minister) has been appointed a member and the chairman of the Medical Board constituted in pursuance of the provisions of *The Workers' Compensation (Lead Poisoning, Mount Isa) Acts*, 1933 to 1945, of Queensland.

Nominations and Elections.

THE undermentioned have been elected as members of the *New South Wales Branch of the British Medical Association:* Abram, Victor Geoffrey, M.B., B.S., 1953 (Univ. Sydney); Bowker, Joan Helen, M.B., B.S., 1953 (Univ. Sydney); *Dawson, Leonard William George, M.B., B.S., 1953 (Univ. Sydney); Price, James Arthur Harold, M.B., B.S., 1953 (Univ. Sydney); Vidor, George Ivan, M.B., B.S., 1953 (Univ. Sydney); Bluett, Kenneth Gabriel, M.B., B.S., 1948 (Univ. Sydney); Borger, James Paul, M.B., B.S., 1952 (Univ. Sydney); Bushell, Douglas Ian, M.B., B.S., 1952 (Univ. Sydney); Connelley, Thomas Joseph Bede, M.B., B.S., 1952* (Univ. Sydney); Harden, William George, M.B., B.S., 1950 (Univ. Sydney); Pepper, Arthur Cecil, M.B., B.S., 1952 (Univ. Sydney); *Warhaftig, Hirsh George*, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act*, 1938-1950.

Diary for the Month.

JAN. 5.—New South Wales Branch, B.M.A.: Council Quarterly.
JAN. 6.—Western Australian Branch, B.M.A.: Council Meeting.
JAN. 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

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